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FUNCTIONAL AND PSYCHIC ELEMENTS IN HEART DISEASE

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The marked increase in the incidence of coronary thrombosis has struck fear into the hearts of men, and has taken a firm grip on their imagination. This dual effect has prepared much fertile soil for cardiac neurosis. The recent publications in the lay press on coronary thrombosis, together with the details of the case relating to the President of the United States, have added fuel to the fire.

Cardio-psychic aberrations have become as great a problem in civilian life as was the case in military practice, and an accurate diagnosis is of importance for the following reasons:

1. They are in themselves partially or completely incapacitating conditions.
2. It is essential to distinguish them from organic heart-disease.
3. Proper treatment is necessary, but often neglected.

The importance of differentiating functional from organic heart-disease must be readily appreciated. The absence of cardiac enlargement, organic murmurs, cardiac failure, angina pectoris, abnormal cardiac silhouette on X-ray, or ECG changes, would indicate that organic heart-disease is not responsible for the patient's symptoms or incapacity.

The diagnosis of functional heart-disease depends upon the combination of a cortico-hypothalamic imbalance in association with a cardio-respiratory functional disorder, which usually occurs after prolonged periods of mental strain or exhaustion, in persons with a positive hereditary history of nervousness or emotional instability. Psychological or psychiatric factors may profoundly influence the cardio-respiratory system through the autonomic nervous system. The stimulus is emotional, and appears to act on the central vegetative nuclei in the region of the hypothalamus. The following is the physiological explanation for these phenomena: Sympathetic or adrenergic activity may cause palpitation by accelerating the pulse, elevating the blood pressure, and strengthening the heart beat. Parasympathetic or cholinergic activity may induce syncope by retarding the pulse, lowering the blood pressure, and weakening the heart beat. The cortico-hypothalamic imbalance may manifest itself by the following symptoms: Blunting of mental acumen for rapid thinking or calculating, lapses of memory, phases of indecision, irritability or depression.

The most significant signs of functional heart-disease are found in the response of the cardio-vascular and respiratory systems to exercise or emotion. Slight exercise increases the pulse and respiratory rates and deepens the respiratory excursion, whereas during sleep or at rest the pulse and respiratory rates are normal. After exercise, the pulse and respiratory rates and elevated blood pressure take a considerably longer time to return to normal levels in patients with functional heart-disease than in normal persons. Systolic murmurs which may occur are usually inconstant, and vary in intensity with change of posture. Cardiac murmurs of this type, unaccompanied by any evidence of structural disease, are not indicative of organic heart-disease.

The symptomatology of patients with functional heart-disease includes dyspnoea, palpitations, left inframammary pain, fatigue, sweating, giddiness, tremors, hyperthermia, anorexia, loss of weight. The signs which may accompany these symptoms are tachycardia, hypertension, and a systolic murmur at the apex or base of the heart.

Although these symptoms and signs may occur as a physiological syndrome of effort in a healthy person who exercises sufficiently strenuously, they are produced more easily, and last considerably longer, in patients suffering from functional heart-disease. Amongst those who display these symptoms is a very definite group who are suffering from chronic infections, such as pulmonary tuberculosis, infection of the sinuses, infection of the gall-bladder or bowel, infection of the genito-urinary tract, or other foci of infection. Another group are patients convalescing from acute infections. Hyper-

thyroidism is sometimes suspected in these cases. These groups are, however, usually distinguishable on clinical, radiological or laboratory findings.

It is well recognized that functional heart-disease may be superimposed on organic heart-disease, but this aspect of the subject is not dealt with in this paper.

TREATMENT

Uneconomic cell-function is one of the most potent sources of ill-health, and the heart is especially prone to respond to stimuli with reckless haste and undue energy, regardless of its own urgent need for rest.

The direction of function along paths of moderation should therefore be our objective. Because of the intense fear associated with heart disease amongst laymen it is essential to treat these patients with the utmost care and tact. Patients with functional heart-disease may need constant reassurance and encouragement to carry on.

Apart from this assurance it is well to try and disentangle any disturbing psychological factors that may be predisposing to or precipitating the symptoms. In some cases a short period of rest may be needed, but this should be followed as soon as possible by a programme of reassurance, re-education and rehabilitation. In the more resistant cases a course of psychotherapy may be required, as adequacy of psychological control is necessary. A full explanation of the condition, and a clear outline of the treatment should be imparted to the patient as soon as a definite diagnosis has been established. It should be realized that a careless disregard of the disorder at the outset may reduce able-bodied men to restricted lives of misery, through fear of sudden death, thus producing chronic invalidism in patients who might otherwise be restored to good health.

In conclusion I would, for the sake of the patient, appeal to our medical colleagues to cooperate with each other in treating a case of functional heart-disease. There is nothing more disconcerting to such a patient, than to be told by another doctor that there is a difference of opinion in regard to his heart condition. Before making such an assertion, it is advisable to discuss the case with the colleague concerned, and so avoid additional anxiety and suffering for the patient.

SUMMARY

1. The increasing incidence of coronary thrombosis has brought with it a spate of cases of functional heart-disease.

2. The physiology of emotional stimuli in relation to the cardio-respiratory system is considered.

3. The importance of differentiating functional heart-disease from organic heart-disease is stressed.

4. The treatment of functional heart-disease is outlined.

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CONFERENCE ON RHEUMATIC DISEASES

The 111th International Conference on Rheumatic Diseases will be held at Aix-les-Bains, France, from 28 June to 1 July 1956. The subject of the conference will be Non-articular Rheumatism and the Problems of the Surgery of Rheumatism, thus completing the series begun by the conference of 1934, which dealt with Inflammatory Rheumatism, and the conference of 1948, where the subject was Degenerative Rheumatism.

Professor Justin-Besançon, of the University of Paris, will preside over the conference, and the general secretaries are Dr. J. Graber-Duvernay, section of medicine, and Dr. J. J. Herbert, section of surgery.

The subjects will include:

Medicine: the extra-articular manifestations of the various forms of rheumatism and gout; the collagen diseases and muscular rheumatism; neuralgia, including lumbo-sciatic; peri-arthritis; rheumatic pathology of adipose tissue; ocular and gall-bladder troubles in chronic rheumatism; acroparaesthesia; psychosomatic association; skin manifestations; biological and chemical reactions; medical, orthopaedic, physical and spa therapy.

Surgery: Post-traumatic necrosis of the hip; pathogenesis of coxarthrosis; osteochondritis; surgical and orthopaedic treatment; coxitis and coxarthrosis; peri-arthritis of the shoulder, sciatica, and their sequelae; ankylosing spondylarthritis; re-education of the vertebral column; post-operative re-education of the limbs.

Persons desiring to present a paper on any of these subjects should forward their MSS before 1 February 1956 to Secretariat de Congrès, Conférence internationale des Maladies rhumatismales, Boîte postale 51, Aix-les-Bains (Savoie), France, where all correspondence should be addressed.

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EDITORIAL

THE SURGEON AND THE SWAB

Many doctors, and surgeons in particular, seem to live in dread of the law. Why this attitude should exist among members of a liberal profession is difficult to explain. It can only be because of the failure to understand that the basic function of the law is to protect the individual's rights. The law as such is impartial and objective, and its protection naturally extends to the doctor as a member of society, as well as to any other individual.

In his professional activities the doctor establishes a legal relationship on every occasion he attends a patient. That relationship is covered by two wings of the civil law, contract and delict. On the touch line, so to speak, is a third branch, the criminal law, which regulates the legal relationship between the State and its citizens. It is only if the doctor should commit an offence that a sanction of the criminal law would follow, while a civil claim could only ensue from a breach of contract with the patient or the commission of a delictual act.

The civil claim that doctors most often have to meet is one based on delict. The touchstone of a delictual claim is negligence. Without negligence on the doctor's part no claim could ever be founded.

Before undertaking any medical procedure the practitioner must be competent to exercise reasonable skill; not that of the highest possible ability but the skill of the ordinary average member of the particular branch of the profession. Failure to exercise the skill of the average member of any speciality would be tantamount to negligence. Of course any doctor might also be held negligent if he failed to exercise the care of the average reasonable doctor in performing a particular task.

The most difficult problem in all cases is to decide if average reasonable skill was used and reasonable attention given to the patient. A problem which sometimes arises (and which only on the rarest occasion reaches the Courts) is that of the surgeon and the swab. Is the surgeon responsible if a swab is left behind? Is it his duty to account for the swabs or not? This legal conundrum has exercised both South African and

VAN DIE REDAKSIE

DIE CHIRURG EN DIE DEPPER

Baie geheeshere, en veral chirurge, leef oënskynlik in die vrees van die reg. Dit is moeilik om te verklaar waarom so 'n houding onder lede van 'n liberale beroep bestaan. Dit kan slegs toegeskryf word aan die feit dat hulle nie verstaan dat dit die grondslagtelike doel van die reg is om die indiwidu se regte te beskerm nie. Die reg as sulks is onpartydig en objektief, en as 'n lid van die samelewing staan die geneesheer eweveel onder sy beskerming as enige ander indiwidu.

In die beoefening van sy beroep gaan die geneesheer 'n wetlike verhouding aan by elke geleentheid wat hy 'n pasiënt versorg. Daardie verhouding word gedek deur twee vleuels van die burgerlike reg, naamlik kontrak en misdaad. Op die kantlyn, as't ware, staan die derde vertakking, die strafreg, wat die regsverhouding tussen die staat en sy burgers beheer. 'n Sanksie van die strafreg sal volg alleenlik as die dokter 'n misdaad pleeg, terwyl 'n siviele eis slegs kan spruit uit kontrakbreuk teenoor sy pasiënt of uit die pleeg van 'n onregmatige daad.

Die siviele eis wat dokters mees dikwels mee te kampe het is dié wat gebaseer is op 'n onregmatige daad. Die grondslag van 'n onregmatige daad is nalatigheid. Sonder nalatigheid aan die kant van die dokter is daar geen gronde vir 'n eis nie.

Voordat hy enige mediese behandeling onderneem, moet die praktisyn tot redelike vaardigheid bekwaam wees; hy hoef nie die maksimum vermoë te besit nie, maar hy moet wel tot die bekwaamheid van die gewone, gemiddelde lid van die besondere tak van die beroep in staat wees. Versuim om die vaardigheid van 'n gemiddelde lid van sy spesialiteit te beoefen kom op nalatigheid neer. Natuurlik kan enige dokter van nalatigheid beskuldig word as hy nie die redelike sorgvuldigheid van 'n gemiddelde dokter in die uitvoer van 'n besondere taak beoefen nie.

Die moeilikste probleem, by alle gevalle, is om vas te stel of gemiddelde, redelike vaardigheid beoefen was en of die pasiënt redelike aandag geniet het. 'n Probleem wat soms voorkom, en wat slegs as uitsondering die geregshowe bereik, is dié van die snydokter en die depper. Is die chirurg daarvoor verantwoordelik as 'n depper agterbly? Is dit sý plig om rekenskap te gee vir die deppers, of nie? Hierdie regtelike strikvraag het reeds beide Suid-Afrikaanse en Engelse howe besig gehou. Die agterlaat van 'n depper is nie op sigself bewese nalatigheid nie. By hierdie soort saak moet die klaer altyd bewys dat die dokter nalatig was, en die bewyslas rus deurgaans op hom.

English courts. The leaving of a swab does not *per se* establish negligence. This has always to be proved by the plaintiff and the onus remains on him throughout.

An early South African case (van Wyk vs. Lewis 1924 AD at 444) decided that it was the duty of the nursing sister to count the swabs, and not that of the surgeon. Of course the doctor cannot merely dismiss his duty as lightly as all that. He must exercise due care, examine his operation site, and see if there are any swabs *in situ*. He must also assure himself from the sister that the count of swabs and instruments is correct. Only then is he entitled to accept her confirmation.

Since this decision of the Appellate Division surgeons have been well protected by competent theatre sisters.

Whatever the legal niceties are, the surgeon is protected as long as he is not negligent. He must always take all reasonable precautions, and never leave anything to chance. The act of negligence can only be inferred after considering every aspect of the operation. What may well be negligence under one set of facts may be quite excusable under different circumstances.

The English Court of Appeal this year sharply shattered the placid contentment of English surgeons. Urry vs. Bierer—1955 CA; *Brit. Med. J.* (1955) **1**, 796—was a case where a gynaecologist left a swab in his patient's abdominal cavity after performing a Caesarian section. The theatre sister had counted the swabs incorrectly, and the surgeon had relied on this count. But it would appear that the surgeon did not use tapes attached to his packs. The evidence of other medical practitioners was to the effect that packs with tapes were generally used, and that tapes attached to packs were an additional help and precaution to enable the surgeon to remove the packs after operation. The Court held that under these circumstances the surgeon was negligent and awarded damages against him and the nursing home jointly.

This judgment reminds us that other precautions are taken by some operators; for instance, artery forceps may be attached to the loose ends of the pack-tapes, and a small piece of radiopaque metal may be embodied in every swab so that if one is left behind it can be detected by X-ray. However in many hospitals of repute where the theatre is served by an experienced sister and nursing staff these additional precautions are not adopted.

Now it must not be assumed that it is always negligent to leave a swab behind. In some cases, after haemorrhage for example, it may be in the patient's interest to leave a swab *in situ* rather than disturb it, until it can be more safely removed on a later occasion. In other cases the patient's condition may be such that it is essential to close the abdomen or terminate the operation without staying to search for swabs.

It does not necessarily follow that South African

In 'n ou Suid-Afrikaanse hofsaak (van Wyk vs. Lewis 1924 AD, 444) is dit bevind dat dit die taak van die pleegsuster is om die deppers te tel en nie die chirurg s'n nie. Maar natuurlik kan die dokter nie so maklik sy plig van hom afskuif nie. Hy moet die nodige sorg dra, hy moet die operasie-setel ondersoek en kyk of daar enige deppers *in situ* is. Hy moet homself daarvan oortuig dat die suster se telling van deppers en instrumente korrek is. Slegs dan is hy daarop geregtig om haar bevestiging te aanvaar.

Sedert hierdie beslissing van die Appèlhof was chirurge nog altyd goed beskerm deur bekwame teatersusters.

Wat die wettiese noukeurighede ook al mag wees, is die chirurg beskerm solank hy nie nalatig is nie. Hy moet ten alle tye alle redelike voorsorgsmaatreëls tref, en moet hom nooit op die toeval verlaat nie. 'n Daad kan slegs nalatig bevind word nadat alle aspekte van die operasie oorweeg is. 'n Daad wat onder een stel feite maklik as nalatig beskou kan word, mag heeltemal verskoonbaar wees onder 'n ander toedrag van sake.

Gedurende die loop van die jaar het die Engelse Appèlhof die rustige tevredenheid van Engelse chirurge rof onderbreek. Die saak Urry vs. Bierer—1955 CA; *Brit. Med. J.* (1955): **1**, 796—is voor die hof gebring omdat die chirurg 'n depper in sy pasiënt se buikholte laat bly het nadat hy 'n keisersnee uitgevoer het. Die teatersuster het die deppers verkeerd getel, en die chirurg het hom op hierdie telling verlaat. Maar dit het toe aan die dag gekom dat die chirurg nie die soort deppers wat bandjies het gebruik het nie. Die getuienis van ander mediese praktisyns was dat deppers *met* bandjies algemeen gebruik word, en dat die bandjies aan die gaasdoeke 'n addisionele hulp en voorsorgsmaatreël is om die chirurg in staat te stel om al die deppers na die operasie te verwyder. Die hof het beslis dat die chirurg hom onder die omstandighede aan nalatigheid skuldig gemaak het en het skadevergoeding teen hom en die verpleeginrigting gesamentlik toegeken.

Hierdie uitspraak herinner ons daaraan dat sommige snydokters ander voorsorgsmaatreëls tref; slagaartangetjies word byvoorbeeld aan die los ente van die depperbande vasgemaak, en soms word 'n klein stukkie ondeurstraalbare metaal in elke depper gesit. As 'n depper dus per abuis agtergelaat word, kan dit deur middel van X-strale opgespoor word. In baie hospitale van goeie stand, waar die teater bedien word deur 'n ervare suster en verpleegsters, word hierdie addisionele voorsorgsmaatreëls egter nie toegepas nie.

Dit moet egter nie sonder meer aanvaar word dat dit noodwendig nalatig is om 'n depper binne die pasiënt te laat nie. By sommige gevalle, soos byvoorbeeld na bloedstorting, mag die dokter dit gerade ag, in belang van die pasiënt, om 'n depper *in situ* te laat totdat dit by 'n latere geleentheid veilig verwyder kan word, liewer as om dit dadelik uit te haal. By ander gevalle mag dit weens die pasiënt se toestand noodsaaklik wees om die buik toe te maak of om die operasie te beëindig sonder om nog eers na die deppers te soek.

Dit volg nie dat die Suid-Afrikaanse howe noodwendig tot dieselfde beslissing as die Engelse Appèlhof moet kom nie, maar die Engelse besluit sal gewis baie tel in ons howe.

Die hof lewer 'n uitspraak van nalatigheid slegs as

courts would come to the same decision as the English Court of Appeal has done, but the English decision would weigh heavily with our Courts.

The Courts would only find negligence established if the conduct of the doctor were not that of the average member of his speciality in the particular circumstances of the particular operation. For example, failure to use a new therapy is not negligence, but once it is universally accepted by the profession failure to do so becomes such.

Doctors have difficulty in appreciating why the law is sometimes open. It is true that the principles of our common law are determinable, but it is seldom found that the facts of one case tally exactly with another. As there is clinical variation in the same disease-process in different individuals, so there is variation in the facts of each legal problem. The medical profession will be the first to admit the enormous changes in the profession, in administration, in control, in the nature of practice, and in treatment, during the last decade or so. The law must obviously lag behind, until each new set of circumstances is tested against legal principle. This can only be done in the Courts, which are the laboratories for legal process. Happily for medical men such experimentation is rare.

die aksie van die dokter anders is as wat verwag kan word van 'n gemiddelde lid van sy spesialiteit onder die besondere omstandighede in dieselfde operasie. Versuim om van 'n nuwe terapie gebruik te maak is byvoorbeeld nie nalatigheid nie, maar as daardie wyse van behandeling eers algemeen deur die mediese beroep toegepas word, word versuim om dit te gebruik wel 'n nalatige daad.

Geneeshere vind dit moeilik om te verstaan waarom daar op sekere wetspunte geen beslissing is nie. Dit is wel waar dat die beginsels van ons gemene reg bepaalbaar is, maar dit gebeur selde dat die feite van 'n besondere saak presies ooreenstem met dié van 'n ander. Net soos 'n bepaalde siekteproses klinies van een mens tot 'n ander sal wissel, so ook het elke wetlike probleem sy variasies. Mediese praktisyns sal dadelik toegee dat daar gedurende die laaste tien jaar geweldige veranderings in die beroep gekom het wat betref administrasie, beheer, die aard van praktyk, en behandeling. Dit is begryplik dat die reg altyd agterna sal kom totdat elke nuwe stel feite teen regtelike beginsels getoets word. Dit word alleenlik in die howe—die laboratoriums vir wetlike probleme—gedoen. Dit tref gelukkig vir die mediese beroep dat sulke ‚proefnemings' maar seldsaam is.

RACE AND DISEASE

The multiracial character of the population of South Africa affords almost unique opportunities for research into the cause of disease based on comparisons between one race and another. This kind of research may be epidemiological or clinical or carried out in the laboratory.

The mortality statistics which have for many years been published by the health departments invariably show substantial differences between the respective death rates of Europeans, Indians, Cape Coloured and Bantu. This applies to the death rate from all causes, and still more to the death rates from specific causes of death and to the infant mortality rate. In one of the large cities[1] the diseases which in recent years contributed most to the mortality of the non-Europeans were tuberculosis, the respiratory diseases, and diarrhoeal diseases, from which the death rate in non-Europeans was 8 times, 6 times and 13 times as great as the death rate in Europeans. These 3 causes of death took a comparatively low place amongst the causes of death in Europeans, their position at the top being taken by circulatory diseases and cancer, which contributed most to the mortality of the Europeans. The mortality rate in the Europeans from these two groups of diseases was nearly 1½ times (cancer) and double (circulatory diseases) that obtaining in the non-Europeans. Thus, although the non-Europeans present higher mortality rates than the Europeans, from certain important causes of disease and death the Europeans suffer more than the non-Europeans.

Comparisons between the different non-European groups (Bantu, Indian and Coloured) reveal similar differences between one group and another.

When the causes are closely examined it is found that these racial differences are due not, apparently, to innate biological differences in the races, but to variations in the environmental conditions to which the races are respectively exposed. Amongst these environmental conditions socio-economic differences are the most potent and there are indications, for instance, that when Europeans are exposed to the same environmental conditions as people of other races then their superiority in health and longevity disappear. In other words, the remedy for the bad health conditions obtaining amongst South African non-Europeans is socio-economic uplift.

An interesting example of the opportunities for research that are provided by racial diversity in South Africa is the excessive prevalence of primary cancer of the liver in the Bantu. In these people (as well as certain Oriental races) the incidence of this condition 'is often more than 40 times as frequent as in Western people'.[2] Berman[2] points out that in this phenomenon we are presented with an opportunity for valuable research, which may pass us by as the living conditions of the Bantu increasingly become influenced by Western culture. The evidence at present available suggests that in the causation of this disease environmental conditions play an important part.

In this issue of the *Journal* (page 1151) an abstract is published of a paper by B. Bronte-Stewart *et al.* reporting an investigation carried out by the C.S.I.R. Clinical Nutrition Research Unit at Cape Town University, under the direction of Prof. J. F. Brock, into the racial incidence of coronary disease in Cape Town. Ancel Keys[3] has shown that atherosclerosis, the forerunner of coronary disease, is associated with a high blood-content of cholesterol, and that the incidence of

both high blood-cholesterol and coronary disease is greater in nations and classes whose diet contains a high proportion of fat. These nations and groups are those with the highest average income, and it is deduced that an important cause of coronary disease is a high fat-content in the diet, which is commonest in the well-to-do classes.

In South Africa the highest racial incidence of coronary disease occurs in Europeans and the lowest in Bantu, with the Cape Coloured in between. Bronte-Stewart and his co-workers have found corresponding significant differences in the serum-cholesterol levels in samples from these three racial groups, and a strongly positive, and possibly causal, association of these differences with the consumption of animal fat in the diet. This, they found, did not apply to the consumption of vegetable fat. For this difference no explanation has yet been found.

A significant finding in this investigation is that the serum-cholesterol varied according to income. Of 3 income-groups, the group with the highest income showed the highest serum-cholesterol level, and that with the lowest income the lowest level. Groups on the same economic plane possessed similar serum-cholesterol levels irrespective of their racial origin.

1. Annual Reports, Medical Officer of Health, City of Cape Town.
2. Berman, C. (1955): S. Afr. Med. J., in the press.
3. Keys, A. (1955): *Ibid.*, **29**, 332. Also Editorial, *Ibid.*, **29**, 329.

LIVING ATTENUATED POLIOMYELITIS VIRUS AS AN IMMUNIZING AGENT OF MAN*

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> 'After having spent years striving to be accurate, we must spend as many more in discovering when and how to be inaccurate.'
>
> *Samuel Butler.*†

The problem of poliomyelitis has recently become one of the most difficult subjects about which to lecture. Thanks to newspapers, radio and television, every layman is now an expert on poliomyelitis, and scientists cannot climb into the ivory tower when discussing it. The most intricate scientific problems concerning poliomyelitis are becoming even more popular topics of after-dinner conversation than sport.

PATHOGENESIS OF POLIOMYELITIS

Before attempting to discuss immunization against poliomyelitis, I should like to talk briefly about the disease itself—not the clinical aspects, because I am not qualified—but a few words about pathogenesis. Let us start with a definition. Poliomyelitis is primarily a mild disease of the alimentary tract, mostly asymptomatic, with a very low rate of secondary involvement of the central nervous system. Many of the infrequent paralytic cases recover completely; only a few become permanently affected, and death occurs only in a few exceptional cases. This definition tends to put the disease in its proper scientific perspective, but it is quite understandable that, for parents of a child menaced by paralysis, the disease is an important and dangerous one, and no efforts should be spared to prevent it.

The causative agent of poliomyelitis is, as you know, one of the smallest viruses. To make things more difficult 'viruses defy definition as a biological class,' according to Schlesinger.[1] Even if the broadest characterization of viruses is chosen—that of Luria: 'Viruses are submicroscopic entities, capable of being introduced into specific living cells and of reproducing inside such cells only'[2]—Schlesinger[1] recognizes 'that we fail to detect multiplication, unless the virus is also capable of producing some detectable abnormality in a host organism. This may, however, not be essential to the continuity and survival of the viral species.'

After offering you these two not very encouraging definitions of viruses and of poliomyelitis, the infection itself may be considered. This may sound very elementary, but it is of interest to mention that the paralytic form of the disease in infants has been known for a long time, perhaps as far back as Biblical times, or earlier. According to Paul,[3] the first clear account of the clinical picture of poliomyelitis we owe to a German—Heine—who observed 30 late-paralytic cases and in 1840 published the classic description of what he saw. The epidemic nature of the disease was not generally recognized until 1890, when a Swede—Medin—described outbreaks in his country. Poliomyelitis is sometimes referred to as the Heine-Medin disease, honoring* the names of these two investigators. It was also known in the 19th and early in the 20th century as 'infantile paralysis' because it seemed mostly to affect young infants. The disease is no longer 'infantile paralysis' in most of the countries where it occurs in epidemic form. It has become more and more a disease of older children, adolescents and young adults.

Several facts are known about the epidemiology of the disease. Scientists today agree—and agreement is rare

* Based on lectures delivered during July and August, 1955, before the East African Branch of the British Medical Association, and the Northern Transvaal and Eastern Transvaal Branches of the South African Medical Association.

† Samuel Butler: Selections from the Notebooks, Jonathan Cape, Ltd., London, 1930.

* American spelling is retained in this article.

in the field of poliomyelitis—that the disease is spread by contact. The virus is very hearty and persists everywhere; but no vector, such as a tick or mosquito, plays an important role in the transmission of the disease.

The virus of poliomyelitis enters the body *via* the mouth. This is also a fact on which scientists now agree. It exits *via* the alimentary secretions or excretions.

Phases of Infection. In the light of the newer knowledge about poliomyelitis, Bodian[4] describes 3 phases of the infection which may be distinguished. One is the alimentary. The virus, having entered the body through the mouth, multiplies in certain cells of the upper and lower alimentary tract. From the results of oral administration of attenuated viruses, to be presented later, it will be seen that the number of cells supporting multiplication of the virus is very small indeed. Experimental evidence now indicates that the multiplication of the virus may be associated with lymphoid tissue, viz. the tonsils in the oropharyngeal region and Peyer's patches in the intestinal tract.[5]

The 2nd phase of infection is probably the vascular. Here the virus spreads from the alimentary tract into the regional lymph nodes, then to the blood, from which it may penetrate certain susceptible organs. There is also experimental evidence that the virus may multiply in special tissues, such as brown fat.[6]

The 3rd phase of infection, either experimental or human, is the neural. Here the virus invades the central nervous system, but the route of invasion of the CNS is still little known. It is possible that the virus penetrates from the blood at certain selected areas of the CNS or by spread along nerve fibres. More experimental work has to be done on this problem to give a definite answer, since it is still by no means certain that virus penetrates the spinal cord directly from the blood vessels.

Clinical Categories of Infection. Paralleling these 3 phases of infection with poliomyelitis, there are several clinical categories of infection.[4] To speak of these categories is, of course, to theorize a bit, but they are based on clinical observations carried on for several years, supported by data obtained in chimpanzees.

Probably the most frequent infection is the silent type, consisting only of an alimentary phase. Here, the virus multiplies in the alimentary tract, stimulates an antibody response, and is excreted by the infected person, who remains well.

The 2nd category of infection is the abortive. Here, probably, the alimentary phase is followed by the vascular phase without central-nervous-system involvement. The vascular phase may accompany abortive infection. In the United States during the past 3 years there has been a good deal of study of viremia after the virus was first isolated from human blood by Ward *et al.*[7] and ourselves.[8] Strains of virus almost invariably have been isolated from the blood in abortive cases.[9] Abortive infection may be characterized by slight headache, fever, and perhaps sore throat and general malaise. Virus has not so far been obtained from blood in human paralytic cases, although in experimental animals viremia precedes paralysis.[4]

The 3rd category—the clinical category of infection—is the non-paralytic. There is involvement of the central nervous system below the level of paralysis. This may seem unbelievable, but it has been proven in the laboratory that, before clinical paralysis can be observed in monkeys injected intracerebrally with poliomyelitis virus, more than one-third of the anterior-horn cells of the spinal cord have to be destroyed. So, in non-paralytic infection, there may be some histological involvement of the CNS.

Finally, the 4th category, the most dreaded, is the paralytic infection in which there is massive destruction of anterior-horn cells in the central nervous system.

PROPHYLACTIC IMMUNIZATION

What is the purpose of prophylactic immunization against poliomyelitis? The primary purpose is to prevent the last two categories of illness, namely, the paralytic infection and the non-paralytic infection with involvement of the central nervous system. If viremia could be prevented, obviously a still better prophylactic immunization would be attained. Complete prevention of the alimentary infection is, at present, an impossibility; and to talk about the eradication of poliomyelitis is stark nonsense.

Immunization in Viral Diseases

Before prophylactic immunization is discussed, the general problem of immunization in viral diseases should be briefly considered. There are several types of immunity following a virus infection, and 'virus infection' does not necessarily mean a disease, but may be an asymptomatic infection, as poliomyelitis usually is.

The first type of immunity, which the immunologist aims at duplicating, is the convalescent type which follows either a spontaneous attack of the disease or recovery from a latent infection. It may also follow an intentional exposure to the original agent. The viral diseases of childhood, in general, immunize for the lifetime of the individual. A common practice has been, when one child of a family contracts German measles or even chicken-pox, to expose his siblings so that they may have the disease in childhood, when a relatively uncomplicated course may be expected. Thus, the convalescent type of immunity stems from either unintentional or intentional exposure to the original agent. However, though the viral diseases of childhood usually convey permanent immunity, this is not the case for all viral diseases, the notable exceptions being influenza and the common cold.

The purpose of 'artificial' active immunization is to parallel convalescent immunity; in other words, to reproduce the lasting immunity which follows recovery from the natural disease without the necessity of exposure to the original virulent agent. This can best be accomplished by exposure to the agent in a modified form; that is, as an attenuated live virus.

Attenuated Live Virus

Although many people, both scientists and laymen, have reservations about the widespread use of live virus vaccines in human beings, the fact remains that in the history of preventive medicine two such vaccines have been the outstanding successes—smallpox and yellow fever. It appeared, therefore, to be entirely

logical to attempt immunization against poliomyelitis using live, attenuated virus.

The choice of strains of live poliomyelitis virus to be employed in man presented the problem of setting criteria of attenuation. By attenuation is meant the modification of a virus to such an extent that, when introduced into the original species, it will produce immunity without bringing on an attack of the disease. In veterinary medicine there are numerous attenuated viruses in use today.[10] Obviously they can be used in that field on a larger scale than in human medicine, because testing in the original animal is possible during the process of attenuation. This is impossible in human medicine.

As far as poliomyelitis is concerned, an arbitrary choice had to be made of laboratory tests in experimental animals which would indicate the degree of attenuation of the virus. The most drastic test appeared to be the introduction of the agent directly into the central nervous system of a primate. If a virus injected intracerebrally into a monkey did not produce signs of poliomyelitis, it seemed improbable that the same virus, when fed to human beings, would cause anything beyond a transitory alimentary infection.

The TN Strain (Type 2)

But how to develop attenuated viruses? In the first phase of the work reported here, a virus strain originally isolated from a human being, and thereafter adapted to the CNS of monkeys, was injected into Swiss albino mice. After several blind passages the virus adapted to the latter species. Following ten or so intracerebral passages in mice, the virus was tested in monkeys and found to be avirulent for intracerebrally injected animals, provided it was maintained for passage purposes in white mice. When this so-called TN strain, representing Type-2 poliomyelitis virus, was passaged in the CNS of cotton rats, it became more virulent for monkeys than when it was passaged in mice. Other studies have indicated that the maintenance of poliomyelitis virus in cotton rats results in a strain retaining some pathogenic properties for intracerebrally injected monkeys. The TN strain was tested for its apathogenic properties through the intracerebral and intraspinal inoculation of monkeys (Table I). No paralysis was evident in the

TABLE I. PATHOGENICITY OF TN STRAIN FOR MONKEYS

Route	*Log of PD_{50}/g. of tissue*	*Paralytic Ratio of Monkeys Injected with Dilutions* of Virus*				
		1·0	2·0	3·0	4·0	5·0
IC	4·4	0/13	0/4	0/4	0/4	0/4
IS	3·4	1/12	0/8	0/8	0/8	0/8

* Expressed as logs to the base of 10.

animals injected by the intracerebral route; and only one of 44 animals in which the intraspinal route was used showed paralysis. It is pertinent to note here that workers in the United States have observed that the intraspinal route of inoculation of laboratory animals is more sensitive than the intracerebral.[11]

Plan of Clinical Trials. So, in February 1950, armed with the knowledge that the TN strain was avirulent when introduced directly into the central nervous tissues of monkeys, we undertook a trial in human beings.[12]

Before this and subsequent trials are described, some of the general conditions under which they were conducted should be stated. First, when information obtained on every single individual was of the greatest value, it did not appear justifiable to administer virus to individuals already immune. Therefore, whenever permission was obtained to feed virus to a child or an adult, the blood of the subject was tested for the presence of neutralizing antibodies against the type of virus to be administered, and only those were fed whose blood was devoid of such antibodies.

The route of administration of the virus was limited to the natural one—the oral. The virus was never injected into human subjects, but was usually given in the form of a milk shake, sometimes with raspberry syrup. In several cases the virus was fed in capsules.

Every individual who received virus was under rigid clinical observation from the day of feeding. Temperatures were taken 3 times daily. Neurological examinations were frequent and there was constant supervision. In no instance were signs of clinical disease observed.

After virus was fed, stools were collected and tested to determine whether virus was being excreted. These isolations were at first made only by the intracerebral inoculation of mice; monkey renal-epithelium tissue-culture is now used for Type 1, and mice for Type 2, which is non-cytopathogenic for tissue culture.

At intervals after virus feeding, blood samples were obtained and tested for the presence of neutralizing homotypic antibodies. This has become comparatively simple. Five years ago it was complicated, since monkeys had to be used as test animals. Today a tissue culture procedure is employed in which the inhibition of tissue growth through multiplication of the virus is indicated by the color change of the suspensions in the tubes.[13]

The SM Virus (Type 1)

In addition to the Type-2 TN virus, another strain, this time representing Type 1, was attenuated through passages in laboratory mice.[14] This was called the SM strain, and originated from a mixture of two Type-1 viruses which became modified as the result of prolonged propagation in PRI mice. The SM strain, in the form of tissue culture suspensions, was avirulent when inoculated intracerebrally into monkeys but was still slightly paralytogenic when the intraspinal route was employed (Table II). The first human trials[12,15] comprised the feeding of the TN, Type-2, strain of poliomyelitis. Up to the summer of 1954, 87 persons had

TABLE II. PATHOGENICITY OF SM STRAIN FOR MONKEYS

Route	*Log of TD_{50}/1 ml.*	*Paralytic Ratio of Monkeys Injected with Dilutions* of Virus*				
		Und.	1·0	2·0	3·0	4·0
IC	7·0	0/4	0/4	0/4	0/4	0/4
IS	6·0	2/5	1/5	0/3	0/4	

* Expressed as logs to the base of 10.

been given this virus. The SM strain was fed first to 3 human subjects, all of whom developed homologous antibodies,[16] and had been administered to 20 additional persons by mid-1954. A summary of the trials is given in Table III. Antibody response was good with both

TABLE III. RELATION BETWEEN TYPE OF VIRUS INGESTED, CARRIER STATE AND ANTIBODY RESPONSE IN MAN

Inoculum		*Ratio of Virus Carriers*	*Last Day After Feeding of Virus Isolation from Stool*	*Ratio of Positive Antibody Reactors*
Type	*Range of Logs of PD_{50} or TD_{50}*			
2	3·1—5·8	47/87*	23	86/87†
1	3·2—5·5	23/23*	171	23/23

* Complete absence of clinical signs attributable to ingestion of virus.

† Eight individuals in one trial developed homologous antibodies upon second feeding of virus.

strains. There was, however, a marked difference between the two in the incidence and duration of intestinal carriage of virus. Of those exposed to the TN strain, slightly more than half became carriers for periods up to 23 days; while the SM virus was invariably excreted by all those receiving it, and for much longer time periods (*vide infra*).

Viremia. The problem of viremia has been mentioned before. Sixty-one of the individuals who were fed Type-2 virus (TN strain), and of whom 30 became intestinal carriers (Table IV), were bled every day up to the 12th day after feeding. Blood samples were tested for the

TABLE IV. ABSENCE OF VIREMIA AFTER ORAL ADMINISTRATION OF POLIOMYELITIS VIRUS

Type of Virus Administered	*Schedule of Blood Collections**	*Ratio of Intestinal Carriers*	*Number of Positive Blood Isolations*
2	1—12	30/61	0/61
1	1—8	3/3	0/3

* Days after administration of virus.

presence of virus, with uniformly negative results. The absence of viremia in individuals who received the Type-1, SM strain, is based on a very small number of subjects, and the problem is undergoing further investigation.

Simultaneous Administration of the Viruses and Interference. Since the TN and SM strains elicited antibody responses when administered separately, the decision was recently made to feed these viruses simultaneously. The antibody response (against Types 1 and 2) of 6 children who were fed the two viruses at the same time is presented in Fig. 1. Although all 6 individuals developed neutralizing antibodies against Type-1 virus, only 3 reacted in the same way to the antigenic stimulus of Type-2 virus, 2 (Nos. 2 and 4) failed to develop Type-2 antibody, and 1 (No. 6) almost failed to do so. This puzzling inability of Type-2 virus to elicit antibody in some cases in which it was fed together with Type-1 led to the belief that interference may have occurred betweeen the two viruses. This problem was therefore subjected to a more exact scrutiny (Table V) with the following results: One subject, who received equal

TABLE V. INTERFERENCE BETWEEN TYPE I AND TYPE II VIRUS IN HUMAN INTESTINAL TRACT

Ratio Between Strains Fed Type I : Type II	*No. of Individuals Fed*	*Intestinal Carriage*		*Antibody Response*	
		No. of Individuals	*Type of Virus Excreted*	*No. of Individuals*	*Type of Virus*
1 : (4·7)*	1	1	I	1	I
1 : (4·8)*	2	2	I	2	I
1 : (4·7)*	1	1	I	1	I
1 : (3·7)*	1	1	I	1	I
		1	II	1	II
1 : (3·2)*	1,000	6	I	6	I
	6			4	II
—	(4·1)†	1	0	1	II

* Figure in parenthesis represents the actual amount of TD_{50} of Type I virus fed (in logs to the base of ten).

† Figure in parenthesis represents the actual amount of PD_{50} of Type II virus fed (in logs to the base of ten).

amounts of the two viruses simultaneously, developed antibodies against Type 1 but not against Type 2. The same situation prevailed when the two viruses were fed simultaneously, but with an increasing proportion of Type-2 virus. It was not until the ratio of Type 2 to Type 1 was 100 to 1 that the interference effect was overcome, and the individual receiving the double virus cocktail developed both types of antibodies. That this may have been an exception rather than the rule is indicated by the antibody response of 6 subjects who received a concentration of Type 2 1,000 times greater than of Type 1. Only 4—and possibly only 3—(see Fig. 1)

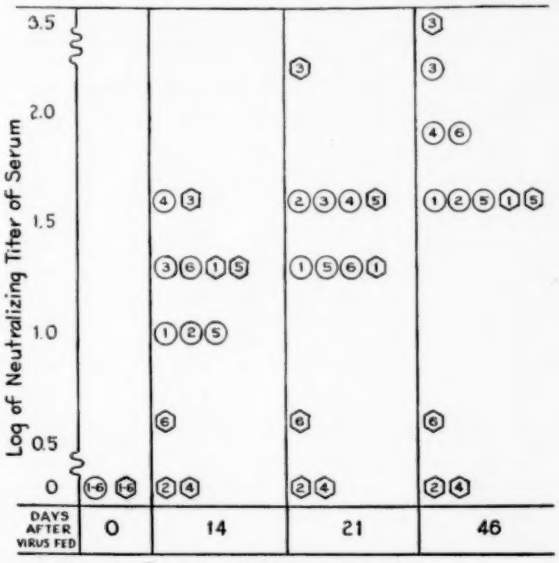

Fig. 1. Antibodies against Types 1 and 2: subjects 1-6.

developed Type-2 antibodies, whereas all developed Type-1 antibodies. The last line of Table V indicates that the Type-2 inoculum possessed good antigenic quality when administered alone. It should be observed, in passing, that the interference phenomenon had to be studied in relation to the presence or absence of antibody response. Data on excretion were not relevant. As has been shown before, and will be mentioned later, Type-2 virus was found only rarely in the stools of subjects fed that virus, and its absence from stools could not be attributed to the interference phenomenon.

The suppression of an agent when it is administered in the presence of another seems to lead to the conclusion that the number of cells of the alimentary tract involved in the multiplication of the two agents is small. Certainly if all the cells of the intestinal mucosa were virus-susceptible, no interference would take place, because a sufficient number of receptive cells would be available for both the viruses to become established. A tempting concept, confirmed recently by Bodian,[5] in studies using chimpanzees, is that multiplication of viruses takes place in lymphoid tissue only—for instance, Peyer's patches. Thus a virus with a short cycle and higher rate of multiplication will dominate, and a slow-moving virus will be suppressed.

Excretion of Type 1 *in Stools and Attempts to Overcome nterference.** Differences were remarked earlier in the carrier rate and duration of fecal excretion of Types 1 and 2. This can best be illustrated with data presented in Table VI. Subjects 13—18 were fed Types 1 and 2 (*vide infra*); subjects 19 and 20 Type 1 alone. It is immediately obvious that subjects 17 and 18 remained intestinal carriers of Type-1 virus—171 and 164 days respectively—for a much longer time than the other subjects. At present, the reason why remains a mystery. No clue is provided by the summary of antibody response in the group which is given in Fig. 2. The antibody response of subject 18 was very sluggish, and the titer of Type-1 antibody obtained on the 42nd day after virus administration

* The results described in this and two subsequent sections were obtained in a trial conducted in cooperation with Drs. George A. Jervis and Joseph A. Stokes, Jr., and Mr. Thomas W. Norton. A detailed account of the trial incorporating all the results will be published elsewhere.

TABLE VI. CONCENTRATION OF TYPE I VIRUS IN STOOLS OF HUMAN SUBJECTS FED SM STRAIN

Pools—Days After Virus Fed	TD_{50} *Titer of Fecal Material from Subjects No.*							
	13*	14*	15*	16*	17*	18*	19†	20†
1—3	6·2	5·7	5·5	5·2	5·5	6·5	4·7	5·7
8—11	6·5			5·7			x0	x0
12—14	5·5	4·5	4·7	4·7	5·7	5·5	x0	x0
15—17	5·7	4·7	4·5				x0	x0
18—20	3·7			5·5	5·2	5·2		
21—24	BP	BP	BP	BP	BP	BP	xx0	xx0
25—28	0	0	x	x0	x0	0	0	0
33—36	0	0	xx0	x4·7	x4·5	3·7	0	0
45—48				xx4·0	xx3·5	3·5		
64—68				3·0	4·7	4·5		
69—82				0	5·4	3·7		
83—92					5·5	BP		
93—102					5·5	0		
103—112					4·4	3·9		
113—120					4·4	3·5		
121—128					3·9	3·6		
129—138					3·4	3·6		
139—148					4·8	3·2		
149—157					4·2	BP		
158—164					0	3·2		
165—171					3·7	0		
172—175					0	0		

BP=Virus isolated on blind passage.
x=Calomel administered
xx=Calomel stopped
* Types 1 and 2 fed.
† Type 1 only.

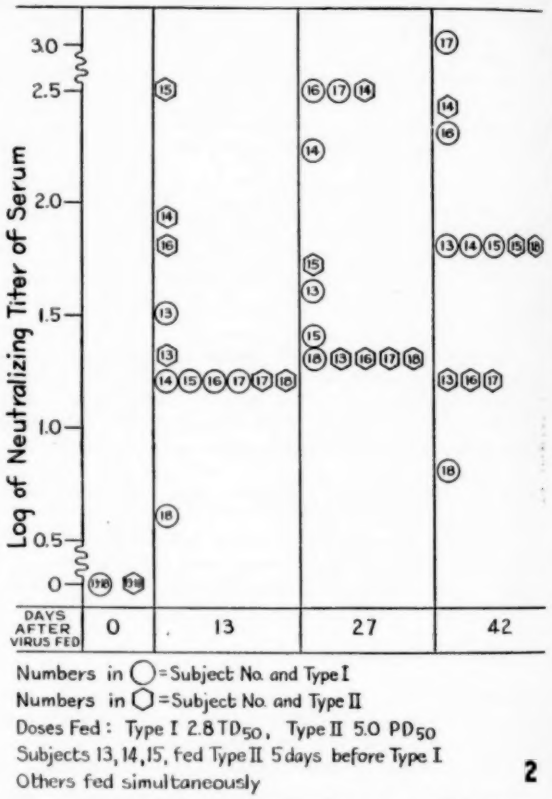

Fig. 2. Antibody titers against Types 1 and 2: subjects 13-18.

remarkably low. In contrast, the Type-1 antibody titer of subject 17 was the highest of the entire group. These seemingly paradoxical observations tend to confirm the opinion that duration of intestinal infection is in no way related to either the presence or level of homotypic antibody in the blood of an exposed individual.

In an attempt to overcome interference, 3 subjects in the same trial (Nos. 13, 14 and 15) received Type-2 TN virus 5 days before Type 1. All 3 developed antibodies against the two types. However, the same response was observed this time in subjects 16, 17 and 18, who received the two strains simultaneously. Although on the basis of these results it would be difficult to conclude that

pre-feeding of Type 2 overcomes interference, examination of a summary of the two trials (Table VII)

TABLE VII. EFFECT OF SEPARATE OR SIMULTANEOUS ORAL ADMINISTRATION OF TYPES I AND II OF VIRUS

Administration of virus	Dose Type 1	Dose Type 2	Subject No.	Duration of Intestinal Carriage*	Antibody Response Type 1	Antibody Response Type 2
Simultaneous	3·2	6·6	1	1—74	+	+
			2	1—82	+	—
			3	1—74	+	+
			4	1—73	+	—
			5	1—53	+	+
			6	1—82	+	—
	2·6		16	(1—25) ‡ (28—68)	+	+
	3·1	5·0	17	(1—25) ‡ (28—102)	+	+
	3·1		18	(1—20) (35—82)	+	±
Separate†	2·8		13	1—25	+	+
	2·8	5·0	14	1—24	+	+
	2·6		15	1—21	+	+

* of Type 1 virus, days after virus administration.
† The TN strain (Type 2) administered five days before Type 1.
‡ Calomel administered on the 25th day.

indicates that, of 9 subjects who received the two viruses simultaneously, 3 failed to develop Type-2 antibody. In contrast, 3 of 3 subjects pre-fed the Type-2 virus developed Type-2 antibodies.

Capsule or liquid? The possibility of bypassing the pharynx as a locus of viral multiplication was investigated by the administration of Type-1 virus (SM strain) in capsule form. Subjects 15, 16 and 20 (Table VIII) received 1 ml. of Type-1 virus suspension mixed with polyethylene glycol in a hard gelatin capsule. Each capsule was carefully washed outside to free it from any extraneous virus contamination. The subject swallowed it, consuming immediately thereafter ½ oz. of milk.

TABLE VIII. COMPARATIVE ADMINISTRATION OF SM VIRUS (TYPE 1) IN LIQUID AND CAPSULE FORM

Virus Administered in Form of	Subject No.	Dose* of Virus Fed TD_{50}	Intestinal Carriage (Days)	Antibody Titer* Days After Virus Fed 13	27	42
Capsule	15	2·6	1—21	1·2	1·4	1·8
	16	2·6	(1—25)(28—41)†	1·2	2·5	2·3
	20	4·7	1—9†	2·4	1·6	1·8
Liquid	13	2·8	1—25	1·5	1·6	1·8
	14	2·8	1—24	1·2	2·2	1·8
	17	3·1	(1—25)(28—41)†	1·2	2·5	3·0
	18	3·1	(1—20)(35—40)	0·6	1·3	0·8
	19	4·9	1—8†	2·4	2·5	2·4

* Expressed as log to the base of 10.
† Calomel administered for control of virus carriage.

From data shown in Table VIII it may be concluded that by-passing the oropharynx failed to have any demonstrable effect on the rate of multiplication of Type-1 virus in the intestinal tract or on the carrier state, as compared to those subjects who received the same amount of virus in milk. Neither was there any difference noted in the homotypic antibody response between the two groups of subjects.

Fig. 3 illustrates the results of another trial of the same kind. Subject 19 received the Type-1 virus (SM strain) in liquid form, and the remaining individuals in a capsule. The antibody response observed for No. 19 on the 42nd day after ingestion of virus was of a somewhat greater magnitude than the titers recorded for Nos.

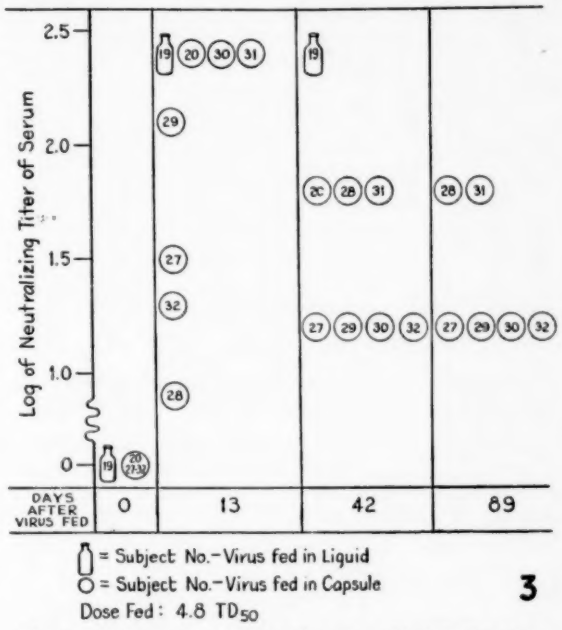

Fig. 3. Antibody titers against Type 1: subject 19 fed virus in liquid; subjects 20 and 27-32 fed virus in capsule.

27-32 (capsule-fed). However, the results for the latter 5 subjects were similar to those shown in Fig. 2 for individuals who received virus in liquid form. The sera of 6 subjects (last column of Fig. 3) after virus feeding by capsule showed no decline in antibody levels as compared with the 42nd-day results.

Attempts to Control the Intestinal Multiplication of the Virus. The high rate and long duration of intestinal carriage following the feeding of Type-1 virus prompted a search for means of suppressing viral excretion. Calomel was a drug of choice, since inorganic and organic mercurials were observed by LoGrippo, Ward and others[17] to be effective in controlling intestinal infection in mice given encephalomyelitis (TO) virus. One organic mercurial compound has also been noted as active against poliomyelitis in the human intestinal tract.[18]

In the initial trial, calomel was administered to 2 subjects 9 days after they received Type-1 virus. The drug was given orally in 10 divided doses per day, totalling 45-70 mg. The results of the preliminary trial were only short of miraculous. Subjects 19 and 20, who were given calomel on the 9th day after virus feeding (Table IX), ceased to excrete Type-1 virus the day after administration of the first dose of calomel, in contrast to subjects Nos. 13 and 18, who excreted the virus for 20-25 days following ingestion. Yet when the same dosage schedule of calomel was applied to subjects Nos. 16 and

TABLE IX. CONCENTRATION OF SM (TYPE 1) VIRUS IN FECAL MATERIAL OF SUBJECTS TREATED WITH CALOMEL AND OF UNTREATED CONTROLS

Stools Collected And Pooled—Days After Virus Fed	TD_{50} Titer of Fecal Material from Subjects Receiving					
	Calomel				Nothing	
	Subj. 19		Subj. 20*		Subj. 13	Subj. 18
	Before†	After†	Before†	After†		
1—3	2·7		3·7		4·2	4·5
4—9	2·7		2·7		4·5	3·2
10		0		0	NT	NT
12—15		0		0	3·5	3·5
16—20		0		0	3·7	3·2
21—25		0		0	1·7	0
26—29		0		0	0	0
30—40		0		0	0	+
40—164					0	+

* Virus administered in gelatin capsule.
† Administration of calomel.
O = No virus isolated on blind passage.

17 (Table X) 25 days after administration of the virus, the results were far from encouraging. Though the virus

TABLE X. EFFECT OF CALOMEL ADMINISTRATION UPON INTESTINAL CARRIAGE OF SM (TYPE 1) VIRUS

Subject No.	Calomel Administration: Days After Virus	Calomel Administration: Mg. Per Day × No. of Days	Intestinal Carriage of Virus: Absence† 1st Noted	Intestinal Carriage of Virus: Duration† of Absence	Intestinal Carriage of Virus: Reappearance† of Virus
19	9—23	45×5 70×9	11	Permanent	—
20*	9—20	45×5 70×6	10	Permanent	—
16*	25—43	45×16	26	26—29	30—43
17	25—43	45×16	26	26—28	28—171

* Virus administered in gelatin capsule.
† Days after administration of virus.

seemed to 'disappear' from the feces for a short period of 2-3 days following the initial dose of calomel, it reappeared promptly in the intestinal excreta and main-

TABLE XI. CONCENTRATION OF TYPE I VIRUS IN STOOLS OF HUMAN SUBJECTS FED THE SM STRAIN OF POLIOMYELITIS

Pools—Days After Virus Fed	TD_{50} Titer of Fecal Material from Subjects Nos.					
	27	28	29	30	31	32
1—3	4·5	4·8	5·7	4·8	Neg.	4·5
4—7	5·2	6·2	5·5	5·5	3·5	4·7
8—11	*3·5	*5·5	*4·5	*3·2	*4·5	
	3·2	5·2	4·8	BP	5·2	*3·2
12—14	Neg.	4·2	Neg.	†Neg.	BP	+
15—17	†Neg.	†+	†Neg.	Neg.	†Neg.	†+
18—20						
21—24	+	+	Neg.	Neg.	Neg.	Neg.
25—28	Neg.	+			Neg.	Neg.
29—32	Neg.	+				Neg.
33—36	Neg.	Neg.				
37—40		BP				
41—44		+				
45—48		+				
49—50		0				

BP = Virus isolated on blind passage.
+ = Virus present on direct inoculation; stool suspension not titrated.
* Calomel administered.
† Calomel stopped.

tained itself for 171 days (subject 17) in spite of prolonged —16 days—administration of the drug. These equivocal effects of calomel were further indicated by the results of a trial in which all 6 subjects (Nos. 27-32, Table XI) received the drug 8 days after virus. Three of the 6 ceased to excrete virus on the 11-14th days after virus feeding, one on the 17th day and one on the 24th; but the other subject continued to excrete virus for 48 days. Since the duration of intestinal infection with poliomyelitis virus varies a good deal, it would be difficult to ascribe any effectiveness to calomel.

*Susceptibility of the Human Intestinal Tract to Infection with Attenuated Virus.** The greater sensitivity of cultures of human epithelial tissues over those of simian origin in the detection of poliomyelitis virus[19] prompted a trial in which serial 10-fold dilutions of viruses of Types 1 and 2 were administered orally to non-immune subjects. The amount of virus fed was expressed in terms of number of plaque-forming particles (on monolayer of monkey renal-epithelium plate-culture) for Type 1,[20] and in mouse PD_{50} for Type 2, which is non-cytopathogenic.

It may be observed from the results summarized in Table XII that the human intestinal tract seems to be susceptible to as small a dose of Type 1 as two plaque-

TABLE XII. SUSCEPTIBILITY OF MAN TO INAPPARENT INFECTION WITH ATTENUATED LIVE POLIOMYELITIS VIRUS

Type I			Type II		
Amount* PFP Fed	Carrier Rate	Antibody Response	Amount† PD_{50} Fed	Carrier Rate	Antibody Response
200	4/4	4/4	45,000	0/3	3/3
20	4/4	4/4	4,500	1/3	2/3
2	2/3	2/3	450	1/2	2/2
0·2	0/2	0/2	45	0/2	0/2

* In gelatin capsule; dose expressed as number of plaque forming particles per capsule.
† In milk; dose expressed as mouse paralytic doses.

forming particles (in monkey renal-epithelial monolayers). If human, instead of monkey, tissue plate-cultures had been exposed to the same concentration of virus, the number of plaques might have been greater. Nevertheless, one has to assume that the human intestinal tract, exposed to attenuated poliomyelitis virus administered in a gelatin capsule, is very susceptible to infection by the virus. Similar, although not so great, sensitivity was noted in subjects who received Type-2 virus. Both individuals who were fed 450 PD_{50} of virus became infected, as indicated by antibody response. Two subjects who received 10-fold less virus (45 PD_{50}) failed to develop antibodies. However, mouse titration based on 0·02 ml. of inoculum injected intraspinally is a much less quantitative method of determining virus concentration than the 'plating out' technique,[20] and it is quite possible that correlation between the susceptibility of the human intestinal tract and that of mouse CNS to an attenuated Type-2 virus is even closer than these results show.

Duration of Immunity. The most important advantage of immunization with live viruses is the persistence of

* This section deals with results which constitute a fragment of a large clinical trial conducted in cooperation with Drs. George A. Jervis, Thomas L. Nelson and Karl F. Meyer and Mr. Thomas W. Norton. Complete account of the trial will be published elsewhere.

immunity. This fact has been borne out by numerous instances in both human and veterinary medicine.[10] Favorable results have so far been obtained in the trials with attenuated strains of poliomyelitis virus. Homotypic

TABLE XIII. PERSISTENCE OF NEUTRALIZING ANTIBODIES AGAINST TYPE II POLIOMYELITIS VIRUS IN SERA OF INDIVIDUALS FED TN STRAIN

Individual Number	*Minimum Protective Titer* of Serum Obtained Months† After Administration of Virus*		
2	2·0(11)	2·0(48)	1·8(60)
3	1·6(2)	2·8(47)	1·8(59)
9	2·2(3)	1·6(41)	1·3(53)
16	2·9(12)	2·6(50)	1·8(62)
5	1·9(1)		1·8(60)
7	2·4(1)		1·8(54)
8	2·4(2)		1·2(54)
10	2·4(1)		1·8(52)
12	1·8(15)		1·8(66)
13	1·6(1)		2·4(60)
14	1·8(13)		1·2(54)

* Expressed as logs to base of 10.

† Figures in parentheses refer to number of months after feeding of virus.

antibody levels in 11 subjects who were fed the TN strain (Type-2 virus) remained approximately the same throughout a 5-year observation period (Table XIII). The results obtained with Type-1 virus were also highly encouraging, although the period of observation was much shorter. From data presented in Fig. 4 it may be

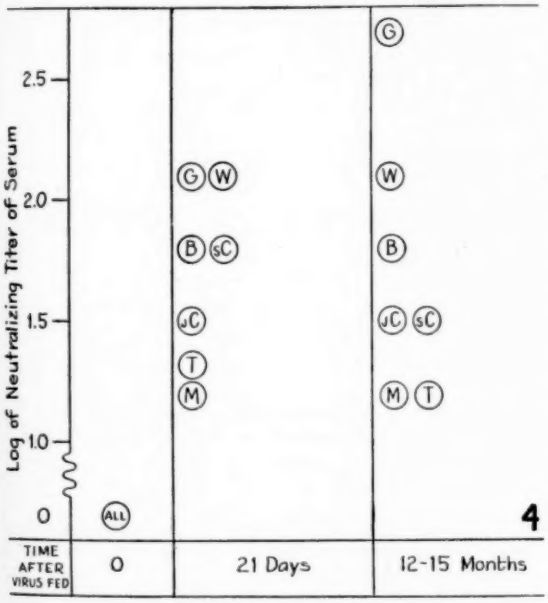

Fig. 4. Persistence of antibodies against Type 1 in sera of children fed living attenuated SM virus (single dose).

noted that none the of 7 subjects showed deterioration in their homotypic antibody levels within 12-15 months following administration of a single dose of SM strain (Type 1) virus. Antibody levels in all of the above individuals will be subjected to frequent scrutiny in the future in the hope that the immunity will be of long duration.

THE FUTURE

The last two tables (Tables XIV and XV) present the author's views on the acceptibility of attenuated viruses as vaccines.

At present, in my opinion, an attenuated virus can be pathogenic for laboratory rodents and still be acceptable, but should not be pathogenic for monkeys injected intracerebrally (Table XIV), and should preferably be non-pathogenic when the intraspinal route of inoculation is employed. As far as I know, however, there are no strains in existence which are consistently non-pathogenic for monkeys injected intraspinally. It might be necessary,

TABLE XIV. ACCEPTABILITY OF ATTENUATED POLIOMYELITIS VIRUS TO BE USED AS LIVING VIRUS VACCINE—RATING ACCORDING TO PROPERTIES OF THE VIRUS

(Personal Credo of the Author)

Animal Host, Tissue Culture, etc., or Characteristics	*Properties of the Virus That Make It:*	
	Acceptable	*Non-Acceptable*
Rodent CNS	Pathogenicity	Immaterial
Cynomolgus CNS: Brain	Non-Pathogenic	Pathogenic
Cord	Non-Pathogenic	Pathogenic
Tissue Culture: Monkey Epithelial or Fibroblast	Preferably Non-Cytopathogenic	Cytopathogenic
Human Epithelial	Preferably Non-Cytopathogenic	Cytopathogenic
Genetic Stability of the Virus	Pure* Clone Desirable	Mixed Population

* No change in the population after passage through human intestinal tract.

therefore, to accept as an immunizing agent a virus which may occasionally produce paralysis in monkeys inoculated by that route. The fact that such strains have been found to be avirulent when injected intraspinally into chimpanzees[22] is comforting, although this again may be related to the rather small number of chimpanzees as compared to monkeys employed in the study.

The choice of non-cytopathogenic strains as preferable for immunization purposes can be explained through the examination of the 3rd line of Table XV concerning fecal excretion of virus. The non-cytopathogenic TN (Type 2) strain is only rarely found in human excreta, and then in concentrations so low that it can hardly be considered contagious. Conversely, the

TABLE XV. ACCEPTABILITY OF ATTENUATED POLIOMYELITIS VIRUS TO BE USED AS LIVING VIRUS VACCINE—RATING ACCORDING TO REACTION OF THE HUMAN HOST

(Personal Credo of the Author)

Sign or Symptom	*Acceptance As A Vaccine*		
	Ideal	*Passable*	*Non-Acceptable*
Clinical Signs	Absent	Absent	Present
Viremia	Absent	Absent	Present
Fecal Excretion of Virus	Absent	Occasional*	Frequent
Antibody Response ..	Good	Good	Occasional

* Depending on whether or not excreted virus is contagious.

cytopathogenic SM strain (Type 1) was found in relatively high concentration in feces, and in some instances excretion of the virus lasted for a long period of time (see above). The characteristic of non-cytopathogenicity possessed by certain strains—i.e., failure to destroy cells of either simian or human origin outside the body—may also condition non-contagiousness.

The homogeneity of the viral population is an essential criterion of attenuation, and methods are now available to determine the purity of a viral strain.[20]

Data presented in Table XV are appurtenant to Table XIV. The reaction of the host should be that of an inapparent, transient infection, followed uniformly by the development of persisting immunity.

The above criteria of 'acceptability' are not absolute; they may have to be changed because of the unavailability of strains which meet the conditions in every respect. Qualifications, determined in a proper scientific fashion, may dictate the approval of different criteria of attenuation.

In our studies during the past 5 years, approximately 200 non-immune individuals have received attenuated strains representing two types of poliomyelitis virus. The stockpile of attenuated viruses fulfilling most of the conditions for acceptance is growing rapidly. The administration of these agents to a non-immune population may, in the not too distant future, bring the realization that the era of permanent immunization against poliomyelitis is not decades away—as originally thought—but relatively near.

This lecture was purposely delivered neither in a euphoric nor in a discouraging tone. I would have liked my listeners to believe everything I said, but I did realize—particularly when I came to revise the talk—that verisimilitude falls short of truth, although the latter no man may ever know.

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AT WHAT AGE SHOULD THE VERY YOUNG MENTAL DEFECTIVE BE SENT TO AN INSTITUTION?

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This question, far from being merely academic, is real and practical. There is a growing tendency to institutionalize very young mental defectives. It was previously shown[1] that in comparison with the trend 20 years ago, there are at present *pro rata* 3 times as many infants under 6 years old being admitted to the Alexandra Institution, one of the 3 large institutions for mental defectives administered by the Health Department of the Union Government. They were being registered at the average rate of about 13 per annum, and at the end of 1954 there were 32 such infants on the register. As no studies have been made of this group, we lack adequate information which can serve as a guide and on which future planning and policy can be based. A survey of these 32 cases is presented as a basic study.

On this question divergent medical opinions are met which deserve examination and which can be presented by means of 3 brief case-studies selected from the 32 as representing as many schools of thought and practice.

I. CASE 1

It is mostly unwise to postpone institutionalization indefinitely, as the history of KN shows. She is a mental defective aged 14 years, with a mental age of 6 years and an IQ of 43 and with a superimposed neuromuscular defect. Her parents are wealthy and have spared no expense in obtaining adequate medical supervision. Since infancy she has been in the hands of a paediatrician, who has taken a keen interest in her, also in complying with the parents' wishes to consult other specialists. Much and varied attention has been focussed on her and every avenue explored except admission to an institution or a special school for mental defectives. The pae-

diatrician and the family doctor would not entertain any such course, despite that the girl is bored, frustrated, unhappy and drifting aimlessly; she is wearing out her mother and is a source of social embarrassment to her elder brother. The family are very attached to her. She dominates them and her parents can go nowhere without her. She is troublesome, always wanting attention from visitors, and speaking to strangers in public.

The optimum age for sending her to an institution has passed, but if she were sent, even at her present age, she could still fit satisfactorily into the new environment with its many occupations and its new friends of her own intellectual status, and where she would be understood and accepted—a life which offers an escape from continuous criticism, from pressure to live up to an unattainable standard, and from the frustration engendered by unequal competition with her intellectual superiors. But the longer this step is postponed the more difficult it will become to sever the existing family bonds. She and her family will be fortunate if they escape a major crisis which will precipitate her committal to an institution under circumstances and at a time when it will be hard for all concerned to adjust to such a life.

We want to guard against suggesting that it is an obligation of all parents of mentally defective children to institutionalize them. One admires the devotion of those who loyally spend many years nursing a mentally defective child, and sometimes even more than one. If, however, they choose this course then, before embarking upon it they should be fully cognizant of its many implications as well as its alternative.

II. CASE 2

To obviate the unfortunate complications of undue postponement another approach, which is responsible for much of the relative increase in the number of infants in institutions, is being tried increasingly. A discussion of this is presented by reference to the case of DH.

When he was born the doctor diagnosed him as a mongol, and took steps to remove him forthwith to an institution, in pursuance of the plan which was originated in America by Aldrich,[2] according to whom the physician must take the lead in precipitating an immediate crisis in order to prevent much more serious difficulties later on. His method of dealing with the arrival of a mongol is *inter alia* to prevent the mother from seeing her baby, so that she won't become attached to him. The physician acts in cooperation with the husband, one or more relatives and a minister of the church. If the husband and close relatives can be induced to accept outside placement, the situation is reported to the mother, who is asked, not to make the decision, but to accept one which has already been made. This was done in the case of DH, who was 7 days old on admission to the institution.

It is a *prima facie* better approach than the previous one, but it is beset with difficulties. For one thing, it is not a simple matter to nurse such young babies in an institution. To the age of 3-7 years the physical care of most—although not all—retarded children is no greater than that of a normal child of the same age, and in this particular case the mother would have proved the best nurse.

Also, it is not clear how the Mental Disorders Act can be interpreted to apply to a child of one week, for on what grounds can one say about any one particular neonate in contradistinction to others that he is 'a person so deeply defective in mind from birth, or from an early age, as to be unable to guard himself against common physical danger'?[3] Nor is he a child in need of care in terms of the Children's Act of 1937.

As it is, one of the doctors who signed the medical certificate erroneously classified him as a mongolian idiot, probably under the false assumption that all mongols are idiots. It so happened that he turned out to be a mongolian imbecile and not an idiot. It may be thought that this is not a serious error, the main issue being that he is truly mentally defective; but it nevertheless remains an error of diagnosis sufficiently serious to land the child in the wrong legal class of defective persons, and in such a grave responsibility as committal to an institution, even slight, inconsequential errors are to be eschewed. Moreover, the diagnostic error could equally easily have been of a different order, with far-reaching, tragic consequences for, in spite of neonatal signs, the child might possibly have turned out to be mentally normal. As Levinson[4] reminds us, 'In cases of mongolism it can be assumed that the baby is mentally defective. One must however differentiate between mongolian defective mental development and Mongoloidism. Mongoloid features even in white babies do not always signify mental deficiency.'

Then again, it is not always possible to differentiate between mongolism and cretinism in the neonate; and yet such differentiation is imperative because mongolism implies a poor prognosis, whereas cretinism responds to treatment. There are as yet no infallible criteria for the differential diagnosis between these two conditions during the neonatal period.

It seems hard on DH that solely because he had the misfortune to be endowed with recognizable mongolian features, the doctor was in a position to anticipate the provisions of the Mental Disorders Act and prematurely dispatch him to the impersonal atmosphere of an institution, depriving him of maternal care and the emotional warmth of a family circle in his first few years of life. Physically he developed satisfactorily in the institution. Few uninformed parents would have observed that he was different from other children.

However, after the committal his parents grew increasingly doubtful about the correctness of the diagnosis. They felt they had rejected their child, and their guilt feelings grew in strength. The mother could not forget her baby, whom she had 'already loved so much' while she was still pregnant and preparing for his arrival. She blamed her misery on her husband and the relationship between the two deteriorated. When DH was a year old she would no longer be restrained and set forth to visit him, expecting to see a monster, because she had misconceptions about mental defect. When he was brought to her she couldn't believe it was her child. There appeared nothing wrong with him, he looked like her other children, and she so persistently refused to part with him that the authorities had no alternative

but to let her take him home with her. The treatment was a failure, and a fresh approach had to be initiated with the family now negatively conditioned to institutions.

In our survey group there were 13 infants whose committal was handled much like that of DH, with minor variations in their ages and in the final outcome. The following common features could be traced throughout their case histories: All were very young, their respective ages on admission being 2, 2, 3, 3, 0, 2, 3, 1, 1, 0, 3, 2, 2 years. Most of them had been on the waiting list since shortly after birth. All were from homes of above-average socio-economic status. In no case was there at the time an emergency family situation which could be resolved only by the removal of the child to an institution. In all cases the mother would have been the more suitable nurse. For a considerable time to come their nursing and care at home would have been no more difficult than that of normal children. In all cases the diagnosis of mental defect was correct, the respective IQs being 13, 12, 14, 53, 50, 22, 58, 37, 46, 17, 16, 28, 21, but none of them was as yet in such special need of care and supervision as can be offered only by an institution for mental defectives. At their respective ages, their presence in the family group could not conceivably have exercised any ill effects on older siblings, nor did they as yet dominate the family scene unduly. Nine had recognizable mongolian features—the only circumstance which rendered their diagnosis at that time possible. Not one of the families was favourably conditioned emotionally for the removal of the child.

Clearly the best time for their institutionalization had not yet arrived, and the details of their committal could have been handled differently, as the following case-study illustrates.

III. CASE 3

JG was also diagnosed as a mongol shortly after her birth, but the physician withheld this information from the parents until several weeks had elapsed. Then he exposed the implications of the diagnosis—that she would not grow up mentally to the same level as other children, she would be unable to work her way through a normal school, she would need a special environment suited to her individual requirements where she would be understood and spared the frustration engendered by unequal competition with other children. During ensuing discussions the physician aimed at keeping the idea of eventual institutionalization active in the planning of the parents. Superficially and outwardly she grew up in her home environment much like a normal child. She was a favourite both at home and in the neighbourhood, and was not an embarrassment to the older siblings. Although the physician had warned the mother that JG would be more prone to illness than her other children, there were no special nursing problems involved. The psychiatric social worker whose cooperation the physician had enlisted kept the parents fully aware that JG was in many respects very retarded mentally, and emotionally they accepted the idea of eventual institutionalization.

It was agreed by all that when the other children of the neighbourhood go to school at the age of 7 years, she would go to 'boarding school'. The parents had been prepared for it and accepted that she would not be eligible for the special classes of the Cape Education Department, but they were comforted by the thought that she could attend the sub-special class in the institution, where she would learn as much as her intellectual capacity permitted.

It was also a great comfort to them that she would have a home and permanent care in the event of their death. Accompanied by the social worker they had meanwhile visited the institution. At first they did not like all they saw, but accepted the reassurance that, although certain aspects of such an institution appeared distressing to normal adults, they did not matter to the mental defective, and had no significance for him. By reserving accommodation well in advance they averted the frustration caused by a refusal of accommodation.

The child and her mother wept a little on parting the first morning, but they had been adequately prepared for the separation. JG went home for week-ends and school holidays and remained the happy child she had always been. She did not remain at home long enough to dominate the family scene. In nearly all homes there comes a time when siblings or parents and their children have to part. In this case everyone accepted the normal school-going age as a natural time for separation. The distress on parting was no more painful than that which arises when normal children go to school, to boarding school, or to employment away from home. The committal was capably handled, she was sent to the institution at a favourable and inconspicuous time, and the emotional adjustment of all concerned was as good as it could be.

IV

On this page 13 children are mentioned, whose committal was handled on the same lines as that of case 2. The other 19 children in the survey group of 32 were of a different order and could not be treated along similar lines. Seven were illegitimate children, 3 came from bad home-conditions, 2 could not be lived with, one had been hospitalized for several years, while 6 dominated the family scene unduly. The common factors governing their removal to an institution were their unquestionably low intelligence, their low socio-economic status, and their dire need of a suitable environment and of better care.

The 7 illegitimate children had previously been cared for by social-welfare agencies or relatives who generally keep such children so long as they can cope with them. At the time of admission their respective IQs were 23, 40, 40, 2, 2, 8, 23, and their ages 3, 1, 4, 2, 1, 3, 3 years. In all cases the intelligence was so low that it was the duty of the State to care for them eventually in an institution. Obviously such children have to be committed early in life, as soon as it becomes difficult for the temporary foster-parents to cope with them, and this appears to be within the first 4 years of life.

Although their parents were still alive, the 3 infants

with bad home-conditions would be designated as orphans in terms of the Children's Act of 1937. Their IQs were 42, 30, 15 respectively, and their ages 3, 2, 2 years. Their case histories leave no doubt that because of parental neglect, alcoholism, desertion and poverty they were in need of protection and it was the responsibility of the State to take over, the only place for them being an institution for mental defectives, and the earlier they could be committed the better.

Of the 2 who could not be lived with, one was a monstrosity whose mother could therefore not accept him emotionally, and the doctor rightly arranged for his committal 2 weeks after birth. The other one was abnormally aggressive, restless, noisy, destructive and unmanageable. He was 5 years 7 months old on admission. Their IQs were 55 and 26 respectively. An institution was the proper place for them and both came when their parents justifiably admitted that they could not live with them.

Another child was 5 years 7 months old and had an IQ of 5 when brought to the institution from the City Hospital, Cape Town, where he had been most of his life. He was deaf, blind, paraplegic, and nearly wholly unresponsive to external sensory stimuli, and he needed proper hospital care. Rare as they are, such cases should be institutionalized as soon as parents or welfare agencies become incapable of coping with them.

The 6 who dominated the family scene were aged 3, 4, 2, 2, 3, 3 years respectively on admission and had IQs of 18, 21, 20, 15, 22, 18. They were helpless idiots who had all along been nursed like babies, but a situation had arisen in the home which made it impossible for the mother to nurse them any longer; she had just given birth to another baby, or she was expecting the next one, or she was ill in bed, or she had to go out to work as the sole support of the family, and in all cases the socio-economic status of the family was low. Usually such cases are in the hands of social-welfare agencies, who make urgent, strong appeals for their committal, and the State has no alternative but to take over when the child's presence in the home has become impossible. The home conditions indicate when the time for institutionalization has arrived, which appears to be from the 2nd to the 4th year of life.

V. CONCLUSION

The modern trend in South Africa to institutionalize very young mental defectives prevails also in the United States and it is remarkable that the relative increase in the two countries has much the same magnitude.

In our country the operative factors have been: (1) Removal of much of the old prejudice against institutions for mental defectives and, as a corollary, a more positive and more enlightened attitude to them, which gradually evolved after their transfer to the Union Health Department from the Department of the Interior and its associations with the Prisons Department; (2) earlier recognition of mental defect following on refinements in diagnosis; (3) the survival of more low-grade idiots consequent on progress in medical science; and (4) post-war expansion of social-welfare services, clinics and mental-health services, and their role in leading parents to institutionalize their pre-school mentally-defective children, especially among people with low socio-economic status.

In consequence waiting lists have grown. In the Alexandra Institution alone there are more than 300 waiting for beds, some of whom are tragically disabled. A re-evaluation of the tendency to institutionalize very young infants is therefore desirable, and the examination of our case histories has suggested broad guiding principles.

No inflexible rules can be framed. There is much in favour of the suggestion to send the mentally defective child to an institution when it reaches the age of 6-7 years, when normal children also go to school for the first time. This leaves sufficient time for the parents to prepare themselves emotionally for the separation. Once it is acknowledged that a child will have to go to an institution, then this step should not be delayed unduly, and particularly not beyond adolescence, if the relatives are to be spared much and often prolonged unhappiness in the home.

If the home conditions are acceptable, it is rarely necessary to remove the child during its first 1-6 years, unless severe epilepsy or specific physical defects of an extremely disabling nature are superimposed, or unless it is so deeply defective as to need constant medical supervision in a hospital.

VI. SUMMARY

A survey is made of 32 mental-defective infants to find an answer to the question: What is the best age at which to institutionalize very young children?

We conclude that while inflexible rules cannot be laid down, broad guiding principles can be suggested. Each individual case is unique, we have to search for its optimum time, and the final recommendation for it will depend on all the evidence.

Although this is seldom the best time, committal can take place quite favourably until as late as adolescence; but it is generally unwise to postpone it later than this stage.

Although the age of 7 will be the best time for many children, there frequently are socio-economic conditions under which it becomes necessary to commit children earlier and even soon after birth, when they are in dire need of proper care.

The underlying factors of the modern trend to institutionalize infants, especially neonates, are briefly noted.

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TWO CASES OF SPINDLE-CELL SARCOMA OF THE RECTUM

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The object of this paper is to place on record two more examples of this rather rare condition, with a brief discussion of its clinical significance and treatment.

Case 1

A male European aged 34 was referred to the out-patient department, Groote Schuur Hospital, on 31 March 1954, complaining of pain on defaecation and the passage of blood and mucus.

He gave a 6 weeks' history of these symptoms, which had been treated by his doctor with suppositories and analgesics to no avail. He was having an increasing number of bowel actions with passage of much flatus and a feeling of distension of the lower abdomen.

The week before admission the number of bowel actions was of the order of 10 per diem, consisting largely of mucus streaked with blood.

He further complained of increasing pain on going to stool and also that he could feel a lump obstructing the rectum.

The patient, a young man in obvious distress and unable to walk, was found to have a temperature of 103° F. Local examination revealed an incompletely closed anus, distended by what appeared to be a tumour presenting from the rectum. Owing to pain, digital examination was not possible and therefore a tentative diagnosis of submucous abscess was made.

The next day, 1 April, arrangements were made to drain this under a general anaesthetic. At operation a round swelling was found bulging into the rectum. It was the size of a tennis ball, arising from the posterior wall so close to the anal margin that it partly distended the sphincter. The mucous membrane over it was smooth but attached to the underlying mass. In consistency the swelling was firm, no fluctuation was present, but in one area on its anterior surface softening was found. The softened area readily admitted the exploring finger. Instead of pus, pieces of greyish white tissue were readily obtained and it became obvious that one was dealing with an unusual tumour.

The patient was then admitted to the general ward for further investigation and the material obtained was submitted for histological examination.

The blood picture was found to be normal, as also the plain X-rays of the chest and pelvis. The Frei test was negative. No other cause was found to account for the temperature, which gradually subsided with antibiotics. There was marked local improvement, with cessation of pain, and the patient readily submitted to rectal examinations.

The histological report (Dr. Golda Selzer) reads as follows: 'The specimen consists of tissue weighing 5 g., dark brown, firm but decomposed. Histologically, 90% of tissue shows inflammatory changes—granulation tissue, pus and much necrosis. However, there are cells with a tendency to concentric arrangements in parts, foci where they are spindle-shaped and gradations between these. These appear to be neoplastic but it is difficult to assess their histogenesis. The possibilities are neurofibrosarcoma, leiomyosarcoma or a sacrococcygeal tumour.'

On hearing that further operative treatment would be necessary, the patient insisted on being allowed to return home to settle his affairs.

On readmission on 23 April a further biopsy was taken under general anaesthetic. The histological report (Dr. M. Sacks) reads as follows: 'Shows better preserved tumour-tissue than in the previous biopsy, although large areas of necrosis are still evident. The features are those of a connective-tissue tumour best designated as a neurofibrosarcoma. Mitotic figures are not frequent and blood vessels are reasonably well formed, but the tumour should be regarded as at least locally malignant, and one would certainly not be surprised if this tumour metastasized.'

On 30 April an abdomino-perineal resection was successfully carried out by Mr. George Sacks. At operation no evidence was found of secondaries in the liver or elsewhere.

Report on the specimen (Fig. 1) by Dr. J. Campbell: 'Specimen consists of anus and 35 cm. length of large bowel. One small sessile polyp is present along the course of the colon. Bulging into lowermost part of the rectum is a mass 6·5 cm. in diameter, which shows surface ulceration at its lower end and is seen on section to compress mucosa and muscular wall, although it does not arise in these structures. No enlarged local or distant lymph-nodes were found. The histology (Fig. 2) is that of a well-differentiated spindle-cell sarcoma, best described as a neurofibrosarcoma.'

Fig. 1. Case 1.

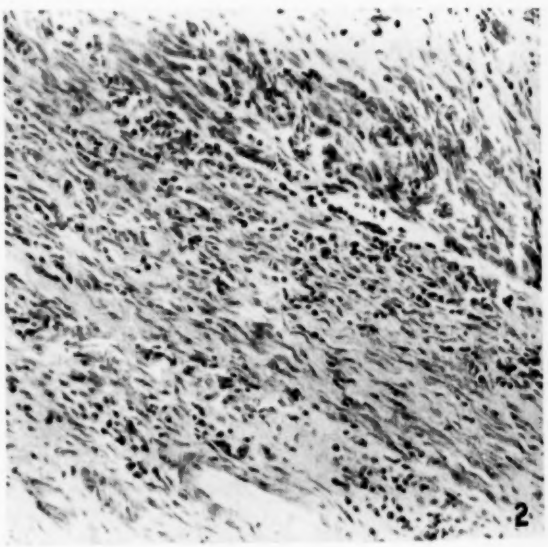

Fig. 2. Case 1.

Convalescence was uneventful and to date, 15 months later, he is well and back at work. He has gained 20 lb. in weight. He has adapted himself well to a 'colostomy life' by simple dietary precautions and enjoys all social engagements.

Case 2

I am indebted to Mr. R. Lane Forsyth for the following notes of a similar case, which occurred in his practice.

A European female aged 52 came for consultation on 27 October 1952, complaining of a 'bearing down' sensation in the rectum. She gave a 3 months' history of her symptoms, which at first consisted of a 'bearing down' sensation in the vagina, which later became transferred to the rectum. There was no alteration of bowel habit and no passage of blood or mucus in the stools. The 'bearing down' sensation had no relation to the act of defaecation.

On rectal examination a firm round tumour, 1 inch in diameter, could be felt low down, arising from the posterior wall. The mucous membrane overlying it was smooth and normal in texture but appeared to be somewhat attached to the tumour. The tumour was sessile, and clinically gave one the impression of being benign, suggesting a tentative diagnosis of a neurofibroma.

On 30 October an excision biopsy was carried out under a general anaesthetic. The histological report (Fig. 3) stated that the tumour was a spindle-cell sarcoma, best described as a neurofibrosarcoma. In view of this report a Miles abdomino-perineal resection of the rectum was carried out on 11 November. At laparotomy no obvious metastases were found but a small nodule the size of a pea was felt on the superior surface of the liver.

The post-operative convalescence was uneventful. Subsequently in the months of January and February 1953 she received a course of deep X-ray therapy to the pelvis through the perineal area.

Thereafter she remained well until March 1954, when she complained of being unwell and on examination was found to have an enlarged nodular liver. Subsequently there was a steady deterioration, with development of ascites, until death supervened on 4 October 1954. The patient therefore survived 2 years from the onset of her symptoms. Autopsy was not performed but it seems justifiable to assume that multiple metastases were the cause of death.

The histology of the 2 cases described was compared and found to be practically identical, viz. spindle-cell sarcomata, probably of neurogenic origin.

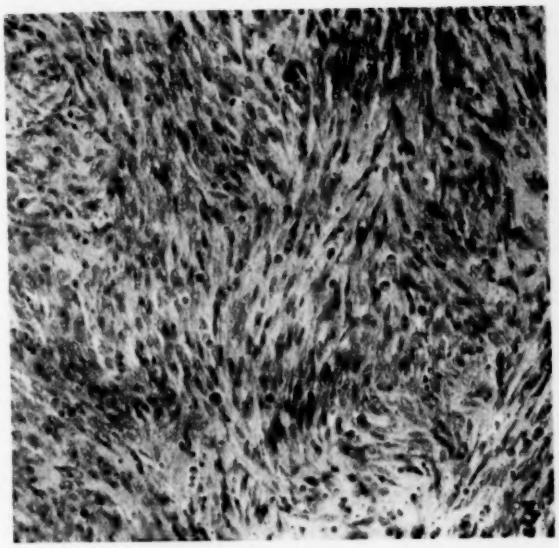

Fig. 3. Case 2.

DISCUSSION

Dukes and Bussey[1] have recently analysed a series of these cases collected from the records of St. Mark's Hospital, London, over a period of 18 years and as an introduction I would like to quote from this authoritative article.

Incidence. Over this period they found 8 cases of sarcoma and 2 cases of melanoma of the rectum, besides 2,200 cases of carcinoma of the rectum examined over the same time. Therefore, of this small series sarcoma and melanoma form roughly 0·5% of malignant tumours of the rectum.

Pathology. Points of distinction between the 3 types of malignant tumours of the rectum are presented by these authors in the following table:

	Carcinoma	*Sarcoma*	*Melanoma*
Site	Anywhere in the rectum.	Anywhere and everywhere in the rectum.	Always ano-rectal region.
Appearance	Most commonly an oval ulcerated tumour with raised edges.	At first a protuberant growth with intact mucosa, ulcerated later.	Dark tumour resembling piles.
Consistence	Feels solid and hard.	Rather soft, resembling brain tissue after fixation.	Firm but not hard.
Cut surface (after formalin fixation)	Yellowish white	Greyish white.	Light to dark brown.

Microscopically, the 8 cases of sarcoma consisted of 4 of lymphosarcoma, 2 of reticulum-cell sarcoma and 2 of spindle-cell sarcoma.

A curious fact was noted in this series that in 4 of the 10 cases more than one malignant tumour was present. In 2 cases (one a lymphosarcoma and the other a reticulum-cell sarcoma) there were multiple tumours present but of the same histology. In the other 2 cases (one a melanoma and the other a lymphosarcoma) there was also present an adenocarcinoma as an independent growth. These writers state, 'We conclude from this experience that a general tendency towards multiple foci of malignancy is a distinguishing characteristic of sarcoma and melanoma of the rectum.'

Prognosis. The same writers state that excision of the rectum has been a successful treatment. The melanoma has the worst prognosis, metastasizing early and running a rapid course. The lymphosarcoma and reticulum-cell sarcoma occupy an intermediate position, while the spindle-cell sarcoma offers the best prognosis, being of a relatively benign nature. On prognosis in spindle-cell sarcoma of the rectum, Dr. Cuthbert Dukes[2] writes as follows:

'I have looked through the records here and I think that there is definitely proof that spindle-cell sarcoma has a good prognosis if treated surgically. The subsequent history of the 2 cases reported in our article was as follows: Case 7 died 8 years and 9 months later having had radium treatment to a tumour of the right thigh. Case 8 died of the operation as reported.

Since this article was written we have had 4 subsequent cases, the details of which are as follows:

1. Synchronous combined excision 2.1.48. Nodule in each lobe of the liver. Died 2 years and 8 months later. Hepatic metastases.

2. Enucleation of tumour 22.11.48 followed by synchronous combined excision for recurrence 23.10.50. Alive and well to date (6½ years).

3. Local removal in fragments 8.6.49. Alive and well to date (6 years).

4. Synchronous combined excision 13.2.50. Died 4 years and 11 months later. Hepatic metastases.

I think you will agree that these results are very satisfactory. If we exclude case 8, who died after operation, only one of the other 5 cases died within 3 years, and 3 cases have lived more than 6 years.'

In comparison with the above cases it is instructive to summarize the series of cases reported by Anderson, Dockerty and Buie[3]:

These writers in their article on myomatous tumours of the rectum report on 10 cases of leiomyosarcoma. The ages varied from 34 to 76 years, the average being 54·5 years. The tumours were situated within 6 inches of the anus, within easy reach of the examining finger or proctoscope, and were situated on either the posterior wall or lateral walls of the rectum, only one being situated on the anterior wall. In size they varied from the smallest (2 cm. in diameter) to the largest (the size of a grape fruit), in-between sizes were described variously in such terms as hen's egg and small orange. They were all sessile growths, soft to firm in consistency. In 5 cases the mucous membrane was intact while in 5 cases ulceration was present. The cut surfaces of the tumours varied from grey to brown.

In none of the cases was a correct pre-operative diagnosis made and the true nature of the tumours was not revealed until the histology became available.

The symptoms varied from the passage of blood, alteration of bowel habit and rectal spasm, to discovery on routine examination.

Unfortunately, precise details are not given in all cases of survival period after operation, but what there is sheds a very interesting light on these tumours.

Two cases were treated primarily by abdomino-perineal resection, in one of which the patient died post-operatively; the result in the other is not specified.

Two cases were treated by local excision plus local radium; both these cases died later from metastases (no period specified).

One case, treated by local excision only, died of metastases (again no period specified).

One case, treated by local excision only, subsequently underwent local excision of recurrences 3 times over a period of 6 years, and finally died after a further recurrence.

One case, treated by local excision and local application of radium, subsequently underwent local excision of recurrences 4 times over a period of 8 years (no further report available).

One case, treated by local excision, subsequently had local removals of recurrence 3 times over a period of 4 years, and then an abdomino-perineal resection for a further recurrence. This patient survived a further 3 years before dying of metastases.

One case, treated by radium only, developed a recurrence 9 months later, for which an abdomino-perineal resection was performed. This patient died 1 year later from metastases.

One case aged 76 and in poor condition was treated by radium only, and died 2 years later from metastases.

These authors further state that over the whole series the average length of life from the date of the first operation was 5·2 years and that 9 out of 10 had died of metastases at the time of writing.

Another point they make is that in this series in no instance did they find lymph-node deposits and that spread was by venous pathways primarily to the liver.

One is impressed by the failure of local excision with or without radium as a treatment in this series (Anderson *et al.*). Further these tumours have an innate tendency to recur locally despite repeated removals over a number of years before finally metastasizing. One cannot but feel, in spite of the melancholy picture presented in this series, that if a radical form of treatment had been adopted primarily, a very different result might have been obtained. It is obvious that a radical abdomino-perineal resection of the rectum offers the best prognosis.

From a clinical point of view the series reveals that the leiomyosarcoma presents itself in the same way as the spindle-cell sarcoma of the rectum. In their general characteristics they are indistinguishable except by histological examination. In particular they both have a latent period during which the growth remains locally malignant before metastasizing. This period, especially in the leiomyosarcoma, may be one of years. It is this feature which helps to make radical surgery a hopeful procedure in these cases. Further from the point of view of prognosis a consideration of the histology is important, e.g. a well differentiated leiomyosarcoma is almost benign in its features and therefore even a cure can be expected with radical surgery.

SUMMARY

1. Two cases of spindle-cell sarcoma of the rectum are reported.

2. Some of the pertinent literature has been quoted from which the following conclusions are drawn:

(*a*) Sarcoma of the rectum is an extremely rare disease, its incidence being approximately 0·5% of all malignant tumours of the rectum.

(*b*) Sarcoma of the rectum presents itself as a round sessile submucous tumour varying in size from a small nodule to a large tumour filling the rectum. Clinically its macroscopic features are benign and to the unwary the small tumour especially lends itself to local excision. Histological examination of such tumours is therefore imperative.

These tumours may present with one or the other of the following: Local pain, rectal spasm or bleeding, change of bowel habit or, as in the one case reported, as a submucous abscess.

(*c*) The differential diagnosis[1] of carcinoma of the rectum should present no difficulty, excepting possibly in the sarcoma which has ulcerated through the mucous membrane.[4] The only serious consideration in a differential diagnosis would be the benign tumours such as a leiomyoma[3] or fibroma, where only the histology would reveal the true state of affairs.

(*d*) Of the various histological types of sarcoma of the rectum the spindle-cell sarcoma and the leiomyosarcoma offer the best prognosis with radical surgery. The records of the results of treatment of these cases by radical surgery are so meagre as to render the question of prognosis a matter of speculation. It would seem reasonable to say that provided surgery is radical and no metastases are present a minimum survival period of 5 years can be hoped for. In favourable cases, where the tumour was discovered early, and histologically was well differentiated, it would not be unreasonable to expect a complete cure.

I wish to thank the following: Mr. Bekker of the Records Department, Groote Schuur Hospital, for his trouble in locating

the necessary case records, Mr. McManus of the Photographic Department, Mr. R. Lane Forsyth for the details of his case, Dr. R. Schapera of the Union Health Laboratories for his co-operation in locating the histological slides of Mr. Forsyth's case, Professor J. G. Thomson and his colleagues of the Pathological Department, Groote Schuur Hospital, for the reports on the histology of the cases reported, Dr. Cuthbert Dukes of St. Mark's Hospital, London, for his friendly interest and cooperation and, finally, Mr. G. Sacks, under whose care case 1 was admitted and Dr. N. Cloete, Medical Superintendent, Groote Schuur Hospital, for their permission to publish the case.

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AN OUTBREAK OF TETANUS

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Large outbreaks of tetanus are comparatively rare in peacetime. In this article an outbreak of 10 cases is described, occuring in Natives as the result of accidental inoculation that took place in the course of 'treatment' by a witch-doctor.

The nature and occurrence of the causative organism, *Clostridium tetani*, determines the incidence and epidemiology of the disease. According to Osler and McCrae[1] the organism is a slender motile anaerobe. It has one end swollen and occupied by a spore and it grows at ordinary temperatures. From two steel nibs dipped in a tetanus culture in 1891 one produced a growth of virulent bacilli in 1902 and the other produced a growth in 1909, which shows its power of survival. It is widely diffused in nature and is a normal inhabitant of the intestines of horses, cattle and other herbivora. Hence it is often found in manure and garden mould as well as in stables, farmyards and places frequented by such animals. Outbreaks of tetanus often occured in the past from the use of gelatin and catgut which had not been properly sterilized. It usually follows some injury, often very trivial, caused by splinters or contaminated by dirt. Tetanus sometimes occurred without any sign of injury, when it was referred to as idiopathic tetanus. Tetanus occurs more commonly in the tropics, where it is a more severe disease, than in temperate climates. Lancaster,[2] of Sydney points out that tetanus has a high incidence in the first year of life, after which it is low until school attendance begins.

These features of the disease have a bearing on the outbreak described here. The first case, a Native woman, L.A., was discovered by the district surgeon, Olifantshoek, C.P., when he was called to a farm in the district on Sunday, 21 March 1954. On examination he found tonic contractions of the neck, back and abdominal muscles, with restricted chest movements and inability to speak or to swallow. There was marked trismus with typical risus sardonicus. Clonic contractions of practically the whole body occurred on the slightest stimulation. Because of the patient's serious condition her back was not examined, but there was a history of an injury of the left knee by a stick. There appeared to be no other injury to the body and the husband, A.A., stated that his wife had no other injury. The patient refused to go to hospital and so she was given the available antitetanus serum (A.T.S.)—6,000 units.

On 30 March the district surgeon was called to another farm, where he found an elderly Native man, W.V., obviously suffering from an attack of severe tetanus. Because of the spasm of the intercostal muscles the patient could not speak to give a history of his illness nor could he cough or expectorate. There was a vertical septic cut wound about 5 inches long on the outer side of his left leg. The history given by relatives indicated that he received the wound in a kraal some time before his illness. His wife denied any other injury. The prognosis was bad and removal to hospital was refused. The patient died about 12 hours later, on 31 March.

On this occasion the first patient, L.A., was again visited and she was found to have improved considerably so that she could swallow with less difficulty. She again refused to go to hospital and another dose of 39,000 units of A.T.S. was administered. On 3 April her husband arrived at the district surgeon's consulting room with a slight trismus and he disclosed the fact that a witch-doctor had treated his wife, himself and the late W.V. He showed a series of small incisions on his back to prove his story and added that the witch-doctor had treated a number of Natives in the district in the same way by making incisions and rubbing in an ointment. He refused to be admitted to hospital and was given 3,000 units of A.T.S. and sent to the police station to repeat his story. Suspicion having been aroused, an exhumation order for W.V. was obtained from the magistrate. On arrival on the farm everyone denied any knowledge of a witch-doctor, but when the coffin was laid bare the wife admitted that W.V. had been treated by the witch-doctor. The body was in an advanced stage of decomposition. On the skin of the back between the scapulae 10 fine horizontal incisions each about ½ inch long were found on each side of the mid-line. Portions of the affected skin were excised and sent for examination at the South African Institute for Medical Research, half placed in preservative, the other half without preservative. *Cl. tetani* was found in these specimens.

On Sunday morning, 4 April, a farmer brought a Native man, W.S., to the district surgeon for treatment. He was found to be suffering from obvious tetanus with continuous clonic and tonic contractions. He was taken to the police station, where he made a statement that he, and the 7 other members of his family, had been

'treated' by the witch-doctor. He was given the last 3,000 units of A.T.S. available.

In the meantime another farmer came with information that 11 of his Natives had been scarified by the witch-doctor. The magistrate then authorised the despatch to the Provincial Hospital, Kimberley, of all the Natives who had received treatment from the witch-doctor. Five of the 24 already showed signs of tetanus at the time of their departure for the hospital, including L.A., A.A. and W.S.

AT THE HOSPITAL

The scene now shifts to the Provincial Hospital, where information had been received of the pending arrival of 24 Natives who were suffering from tetanus or were expected to break out with the disease. They duly arrived on 4 April 1954, about 5 hours after departure, and were immediately sorted into 3 groups, namely group A, of 2 elderly adult Natives with very severe tetanus, group B, of 3 adult Natives with moderate tetanus, and group C, the other 19 Natives, none of whom showed obvious signs of tetanus and which included all the youngest individuals in the party (9 of them under 12 years old). The information that had reached the hospital was to the effect that infection had resulted from rubbing a special ointment into the scarifications made by a witch-doctor, who held out to his patients that his treatment was a panacea which could cure most ills and ranged from prolonging life beyond the natural span to increasing virility. Other evidence was obtained later at the trial of the witch-doctor.

Treatment was started immediately with groups A and B, who received paraldehyde (3 c.c. intravenously and 8 c.c. intramuscularly), A.T.S. (100,000 units intravenously and 100,000 units intramuscularly), penicillin (1 million units), streptomycin (1 g.) and tubocurarin (20 mg. intramuscularly); besides a nutrient rectal drip.

The wounds of all 24 Natives were packed with moist hydrogen peroxide dressings without active removal of the scales. The wounds were the result of the witch-doctor's cut on various parts of the surface of the body.

Those in group C, in addition to dressings of the wounds, were given A.T.S. intramuscularly; adults 10,000 units and children 3,000 units.

During the first 4 days 4 persons in group C showed clinical signs of tetanus in the form of a moderate degree of trismus. On discovery these 4 patients were given the same initial treatment as groups A and B. After initial treatment the 9 cases of tetanus in hospital received the following daily treatment until symptoms disappeared: crysticillin (2 c.c.), streptomycin (1 g.), tubocurarin in oil (20 mg.), and sedation according to needs. Dressings of hydrogen peroxide were continued until the wounds were clean and healing.

A case in group A—W.S., an elderly man—died the day after admission, while all the others recovered within 3 weeks without further incident or complication. None of the cases received intravenous pentothal drips, nor was any lumbar puncture done or any A.T.S. given intrathecally. The most serious cases occurred in adults and the 2 cases that died were elderly debilitated males.

THE WITCH-DOCTOR

While these cases were in hospital the police had traced the witch-doctor, (S.M.) a Native labourer aged 40 years and apprehended him on 7 April. When the Natives concerned had all left the hospital they were called as witnesses at the trial (on 5 May) of S.M., who was charged on 2 counts of manslaughter in that he had unlawfully assaulted W.V. and W.S. with a razor blade or similar instrument and inflicted fatal wounds, and on 3 counts of fraud in that he falsely and with the purpose to defraud pretended that he could treat and cure certain of the witnesses of their bewitched state, accepting remuneration for the 'treatment' knowing that he could not cure them or alternatively contravening section 34 (1) of the Medical, Dental and Pharmacy Act, No. 13 of 1928, as amended, in that he unlawfully practised for gain as a medical practitioner not being registered as such.

When the police arrested the accused they found amongst other things 7 bags of Native medicines, 1 tin of ointment, 2 horns with ointment, 3 pocket-knives, 1 table-knife, a razor with a packet of razor blades, a pair of scissors and a bag of witch-doctor's bones. The medicines were sent for examination to the South African Institute for Medical Research and *Clostridium tetani* was isolated from the tin of ointment but from no other medicines.

At the trial the evidence showed that the patients L.A. and A.A. were 'treated' by incision and ointment on 19 March, became ill 3 days later and still showed signs of trismus on 4 April when they were sent to hospital. Patient W.V., who died on 31 March, was 'treated' on 25 March, and W.S., who died in the hospital on 5 April, was treated on 26 March. The incubation period in the case W.V. was 2 days, in the case W.S. 3 days. It also transpired that the witch-doctor S.M. had collected about £14 cash in fees, as well as a pure-white goat valued at £1 10s. 0d., which he had slaughtered, and a mare valued by the owner at £6, which he had collected from a patient who could not pay in cash. The police recovered £5 and the mare from the witch-doctor S.M., who was found guilty and sentenced to 1 year's imprisonment for each of the first 2 counts and to 3 months' imprisonment or a fine of £25 on each of the other 3 counts.

DISCUSSION

It is unusual in peacetime to have to deal suddenly with numbers of cases of tetanus. The method of infection is unusual. A Native labourer, pretending to be able to diagnose, treat and cure a bewitched state, was able to persuade at least 25 people to believe and submit to painful treatment by him. 'Throwing of bones' is a method of diagnosis which impresses the gullible and induces them to believe what they are told by the operator; it is a rite which the uneducated have come to accept as capable of indicating the origin and extent of their ills, fancied or real. The gullibility is not surprising if one remembers the superstitious beliefs on which many educated and cultured people act. The public need to be perpetually protected from exploitation by those who take advantage of superstition. The witch-doctor was quickly traced.

Under the circumstances the death rate was low and those who succumbed had already been weakened by old age. In a discussion of a paper by Brigadier A. Sachs,[3] Harold Edwards stated that some 80 civilians die annually in England, with a case mortality rate of 50%. Slome[4] contrasts the annual death rate from tetanus in England and Wales (1·6 per million) with that in South Africa (16·1 per million), where the tetanus deaths number about 200 per year, and the rate is much higher in the Coloured and Indians than in Natives or Europeans.

As regards passive immunization in treatment Firor[5] recommends an initial intramuscular injection of 50,000 units A.T.S. with subsequent daily injections of 5,000 to 10,000 units. There is always the danger of allergy or anaphylactic shock (Sheldon Swift[6]), but no such reactions were noted in this outbreak; they are rare in Natives.

Owing to pressure of work there was little time to make a detailed analysis of the results of treatment. The value of tubocurarin could be assessed because no controls were used. In no case did respiratory embarrassment result; the dosage given was much smaller than that recommended by Frank Glenn[7], namely 1 to 1·5 mg. of tubocurarin intravenously per kg. body-weight initially, followed by smaller doses.

As regards the treatment of the wound it was considered that, the incisions being superficial, excision would not be justifiable. Hydrogen-peroxide dressings combined with antibiotics gave satisfactory results in combating sepsis and preventing pulmonary complications. No antibiotic has so far proved effective in tetanus.

SUMMARY

Ten cases of tetanus, of which 2 died, are reported. They occurred amongst 25 Natives who were 'treated' by a Native witch-doctor by multiple incisions in the skin and rubbing in of ointment. *Clostridium tetani* was later isolated from a tin of ointment in the possession of this witch-doctor.

The method of treatment before and after admission to hospital are described.

The legal proceedings and results are mentioned briefly.

We are grateful to Mr. F. de Villiers for his encouragement and permission to publish details of the legal proceedings, to Dr. G. de la Bat for permission to publish details of the treatment in hospital, and to the Secretary for Health for permission to publish the article.

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RESEARCH FORUM, UNIVERSITY OF CAPE TOWN

SERUM CHOLESTEROL, THE DIET AND CORONARY HEART-DISEASE

Abstract of Paper by B. Bronte-Stewart, M.D., M.R.C.P., Aileen D. Moodie, A.M.I.A., A. Antonis, Ph.D., L. Eales, M.D., M.R.C.P. and J. F. Brock, D.M., F.R.C.P.*

Epidemiologically coronary heart-disease has certain peculiarities. Over the last few decades, there has been an apparent increase in incidence, affecting, too, younger and younger men. An abrupt drop in mortality from this disease occurred during the war years in certain occupied countries. There are wide inter-racial differences in susceptibility and within a given race the more economically privileged, such as the professional and executive occupational classes are far more prone than the unskilled-labour classes. Any theory of the causation of coronary heart-disease must explain these trends.

In Cape Town there is a wide inter-racial difference in susceptibility to this disease. The European is at one extreme, the Bantu at the other. The Cape Coloured lies in between. The relative immunity of the Bantu is seen throughout the African continent south of the Sahara, and the possible mechanisms underlying this are: (1) A different coronary-artery system, (2) a lessened tendency to intravascular clotting, (3) some difference in cholesterol metabolism that protects the Bantu from coronary heart-disease.

The last-mentioned possibility was investigated in 364 apparently healthy men, of whom 132 were Bantu, 118 Cape Coloured and 114 European, all in the 40-58 age-range. Parallel with our racial experience of coronary heart-disease we found highly significant differences in the mean serum-cholesterol levels, with the Bantu at one extreme (166 mg. per 100 ml.) the European at the other (234 mg. per 100 ml.) and the Cape Coloured in between (204 mg. per 100 ml.).

These racial groups were subdivided on the basis of their monthly money-income into 3 groups, and within each racial group the most economically privileged possessed the highest mean serum-cholesterol levels. Furthermore groups on the same economic plane irrespective of their racial origin possessed similar levels of the serum cholesterol. Undoubtedly racial or genetic factors could not be the sole or even principal explanation.

An attempt was made to correlate many of the environmental factors that accompany economic privilege with these serum-cholesterol trends. The one strongly positive, and possibly causal, association was the dietary animal-fat consumption. This did not apply to the vegetable-fat consumption. This parallelism of trends was retained no matter whether the groups were subdivided on the basis of economic privilege, degree of physical activity in the occupation, or the extent of alcohol consumption.

Diets rich in animal fat, however, possess liberal quantities of animal protein, with resultant vitamin-changes and a decrease in the starch and cellulose content. It became necessary therefore to study the effect of fat feeding on individuals under controlled conditions. In the 4 cases studied (to date) on isocaloric and isonitrogenous diets, animal fats in the form of beef dripping, butter, meat and eggs led to a prompt rise in the serum cholesterol. No such effect was produced by feeding vegetable oils in the form of ground-nut oil and olive oil even though the percentage of calories derived from these oils was suddenly raised from 3% to 45% and even to 67%. A significant finding was that hydrogenated ground-nut fat behaved differently from the non-hydrogenated oil. It became apparent then that the effects of fat feeding on the serum cholesterol may be determined by the fatty-acid

* Read on 12 October 1955.

composition of the fat concerned. Investigations have been in progress to determine the effect of those fatty acids which are destroyed or altered in the hydrogenation process. One of these is linoleic acid, known to be an essential fatty acid having biological properties. This led to the use of sunflower-seed oil, a vegetable oil with a high linoleic-acid content, on an individual whose serum cholesterol had been elevated previously by cholesterin feeding. Despite the continued consumption of cholesterin, a prompt fall in the serum cholesterol followed the addition of sunflower-seed oil to the diet. It was emphasized that other factors may be responsible for these effects and that further work is necessary before conclusions could be drawn.

Finally it was pointed out that although the relationship between the level of the serum cholesterol and the incidence of coronary heart-disease was well established, other factors probably influence the pathogenesis of this disease. Nevertheless two significant facts have emerged from Groen's recent studies in Holland on vegetarian and non-vegetarian monks. The former, at given age, possessed lower serum-cholesterol levels, and there was a far greater proportion of the vegetarian monks who had survived 65 years of age. This appears to lend support to the concept that the nature of the dietary fat adequately explains these epidemiological peculiarities of coronary heart-disease.

COLLEGE OF PHYSICIANS AND SURGEONS OF SOUTH AFRICA : KOLLEGE VAN INTERNISTE EN CHIRURGE VAN SUID-AFRIKA

T. B. McMURRAY, M.Ch.Orth. (L'pool), F.R.C.S. (Edin.)

Hon. Secretary and Accountant, College of Physicians and Surgeons of South Africa

Since its incorporation on 21 July 1955 the College has been open for the admission of Associate Founders for a period of 6 months. This period will end on 17 January 1956, and after that date admission to the College will be by examination only. All those registered medical practitioners in the Union of South Africa who wish to avail themselves of this opportunity of joining the College as Associate Founders must do so before the latter date. Queries from those interested should be addressed to the Secretary of the College of Physicians and Surgeons of South Africa, P.O. Box 120, Cape Town.

Progress of the College

Since the Inaugural Meeting held in Johannesburg in May 1954, the Constitution of the College has been completed and submitted to the Registrar of Companies and the Minister for their approval. This was obtained on 21 July 1955, and from that date the College was approved as a non-profit-making Company.

The original Founder Members and Founder Fellows altered their designation to Founders, and from their deliberations the Constitution was finally decided. It was at the Inaugural Meeting also that it was decided that Associate Founders could join during the 6 months after the registration of the College.

Activities of the College

1. The College will conduct examinations in all branches of postgraduate study similar to those examinations held overseas and will issue diplomata of comparable value.
2. The College will maintain a register of those who have obtained their diplomata, and these examinations will probably have reciprocity with similar examinations overseas.
3. The College will maintain a library and research organization.
4. The College will sponsor postgraduate teaching throughout the Union by lectures and demonstrations conducted by the Members and Fellows of the College.

Organization of the College

The College will be divided into various divisions and faculties corresponding with the specialties in Medicine and Surgery and these will be relatively autonomous. Each faculty and division will provide from amongst their members panels of examiners and also will lay down the standard to be set in the postgraduate examinations of the College.

The Governing Body

The administration of the College is vested in a Council to be elected as soon as possible after 17 January 1956. This Council will consist of 12 members, of whom not more than 5 and not less than 1 will be elected from each of the four Provinces of the Union.

The Council will elect a President, a Vice-President, a Treasurer and a Registrar, and these will form the Executive of the College. In addition, the Council will nominate an Examinations and Credentials Committee, who will be responsible for the administration of examinations; that is to say, they will be in charge of the actual organization of an examination and the provision of material. At the same time, they will be empowered to ascertain whether individual applicants have completed the course of study necessary for entry to the examinations.

The Examinations

It was decided at the Inaugural Meeting that no diploma would be given to Founders or Associate Founders, since that would make it appear that diplomata could be purchased; on the other hand all Founders and Associate Founders will obtain all the privileges of Fellowship of the College and be entitled to take part in all discussions and vote on issues as if they were Fellows of the College by examination. They will not, however, be able to put letters after their names signifying the acquisition of a diploma.

Objects of the Founders

The object of the Founders in sponsoring the College is to build something for the future of South African Medicine. They regard themselves as the trustees of the College whose duty it is to guide it until there are sufficient Fellows who have reached maturity and seniority to take on this charge. The Founders believe sincerely in this object and will welcome any registered medical practitioner who wishes to join with them in completing this work.

Please note the College is only open for admission of Associate Founders until 17 January 1956. Admission to the College as an Associate Founder is by application and an admission fee of £31 10s. 0d. must be paid on joining.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Newport Trading Corporation (Pty.) Ltd., Johannesburg, Sole Agents for the Union of South Africa of Messrs. Knoll A.-G., Chemical Works, Ludwigshafen-on-Rhine, announce the introduction of *Hepsan-'Knoll'*, a new remedy for the treatment of hepatopathies, and submit the following statement:

One Hepsan ampoule of 5 c.c. contains 0·25 g. of the novel choline salt of acetyl-methionine and 1·25 g. of acetyl-methionine-sodium (corresponding to 1·0 g. of methionine and 0·1 g. of choline). The syrup contains 10% choline salt of acetyl-methionine and invert-sugar syrup. One teaspoonful of the syrup, equal to approximately 5 g., contains about 0·25 g. of methionine and 0·20 g. of choline.

Hepsan has a detoxicating action which is due to its reactive sulphur atom; it is lipotropic and promotes circulation in the

liver. Both in clinics and in general practice Hepsan is administered in infectious, dystrophic, and toxic, icteric and anicteric, liver-parenchyma disorders, in fatty liver, in the initial stage of hepatic cirrhosis with fatty infiltration, in threatening hepatic coma, and for liver protection, e.g., before and after narcoses, in severe burns, etc. As a general dose 1 ampoule daily or every other day may be given by slow intravenous injection (at least 5 minutes per ampoule). In acute life-threatening cases a higher dosage (up to 4 ampoules) is advisable. Hepsan Syrup is indicated in chronic, subacute, and acute liver diseases of obvious mild degree. Adults are usually given 1 teaspoonful thrice a day, children half this dose. Regarding tolerability no objections have been reported.

Soventol 'Knoll' (tablets, jelly) is an antihistaminic, containing 0·05 g. of N-phenyl-N-benëyl 1-4-amino-1-methylpiperidine-hydrochloride per tablet. The jelly contains 2% Soventol lactate. Its therapeutic effect is antihistaminic, antipruritic, and antispasmodic. The antihistaminic action in man is reported to last as long as 10 hours.

Indications: pruritus of any aetiology, especially itching dermatoses, allergic reactions caused by histamine or histamine-like substances, reactions of idiosyncrasy, insect bites, motion sickness, and allergic eye diseases; and to cut short an acute cold. For topical treatment of skin diseases the lesions are covered with a thin film of Soventol jelly whereby a pleasant cooling effect is experienced. Owing to the suppression of itching with ensuing omission of scratching irritations, the healing process will be considerably shortened. The sedative action of Soventol, which in some patients may cause drowsiness, is eliminated by the addition of Cyclohexylisopropylmethylamine which also increases the antihistaminic effect; this combination is available as *Soventolettes* and can preferably be used as an antihistaminic for daytime as well as in ambulant practice in all indications of Soventol. The general dosage of Soventol is 1 tablet; it can be doubled, if necessary, and repeated after 6-8 hours. Soventolettes 1-3 dragées *pro dosi*, repeated after about 6 hours, if required. For children correspondingly smaller doses.

Priatan 'Knoll' (tablets, solution, cough syrup), for the treatment of asthma, contains per tablet 0·015 g. of the new 1-dimethyl-phenyl-iminothiazolidine hydrorhodanate, 0·015 g. of 1-ephedrine hydrorhodanate, 0·050 g. of theophylline and 0·400 g. of phenyldimethylpyrazolone. The solution for oral use as well as for inhalation with aerosol apparatus contains 3% theophylline acetate of 1-dimethylphenyl-iminothiazolidine and 3% theophylline acetate of 1-ephedrine. The cough syrup, besides its thiazolidine and ephedrine compounds, contains also various herbal extracts and small quantities of alkali bromides which facilitate expectoration and soothe the irritated respiratory organs.

The action of Priatan is broncho-spasmolytic, anti-oedematous, secretolytic, analgesic, anti-allergic, and anti-histaminic, and increases the vital capacity. No cardiovascular strain, in particular no hypertensive effect. Priatan is indicated in bronchial asthma (also mixed forms with cardiac asthma), status asthmaticus, bronchitis, silicosis, emphysematous dyspnoea, hay-fever, and other allergic diseases. The cough-syrup is used in coughs of all types, whooping cough, bronchitis, etc. The dose is ½-2 tablets, to be taken several times per day, if necessary, or 10-20 drops of the solution in some liquid 3-4 times daily if required, and the same dose in an attack; children ¼-½ as much. The solution can also be used in aerosol therapy; in acute cases the effect is often felt after 2-3 inhalations. The dose of the cough syrup is 1-2 teaspoonsfuls several times daily, children ½-1 teaspoonful 3-5 times a day. Tolerability is reported to be very good.

Spansule Drugs

M. & J. Pharmaceuticals (Pty.) Ltd., Port Elizabeth, are introducing 'Spansule' products, manufactured by Messrs. Smith, Kline and French Int. Co., Philadelphia, USA, and submit the following statement:

Drugs presented in 'Spansule' form are distributed among numerous tiny pellets contained in a capsule. These pellets are so prepared that the active drug is released gradually over a period of 8-10 hours. This method of presenting drugs for oral administration is a major advance in posology. One capsule produces an even and smooth therapeutic effect throughout the day or night.

The method is not to be confused with enteric-coated preparations which merely delay the release of a drug until it reaches the small intestine, when the whole dose is released at once.

With the 'Spansule' presentation a proportion of the drug is released immediately so that it begins to act as soon as it would if given in an ordinary tablet. Thereafter the remainder of the dose is released a little at a time throughout the day. Since the dose is split between so many tiny pellets the therapeutic effect is smoother, less liable to be affected by variations in gastric motility, and therefore more predictable.

With the 'Spansule' presentation one capsule in the morning or at night is all that is required to ensure a sustained uniform level of therapeutic activity throughout the day or night. Apart from its convenience to the patient and easiness to remember, a one-dose-a-day schedule eliminates the variations so often introduced into a t.i.d. scheme by the patient—the missed midday dose and the double dose in the evening to make up.

Dexedrine Spansule

There is at the moment only 'Dexedrine' available in this form but a wide range of drugs is contemplated.

The therapeutic effect is continuous, smooth and sustained. When used in the reduction of weight the control of the appetite is constant and prolonged between meals, as well as at mealtimes. Each 'Dexedrine Spansule' contains 10 mg. or 15 mg. (according to strength) of 'Dexedrine', distributed among many tiny pellets with varying disintegration times.

In the following conditions the recommended dosage is 10-30 mg. of 'Dexedrine' in Spansule form, usually taken on rising or at breakfast: Obesity (in conjunction with a low-calorie diet) depressive states, alcoholism, drug-induced drowsiness. (The effect of one capsule may last as long as 12 hours, and if the dose is not taken early insomnia may ensue).

'Dexedrine Spansule' capsules are available, on prescription only, in bottles of 30 capsules.

MEDICAL DEGREES CONFERRED AT THE UNIVERSITY OF THE WITWATERSRAND

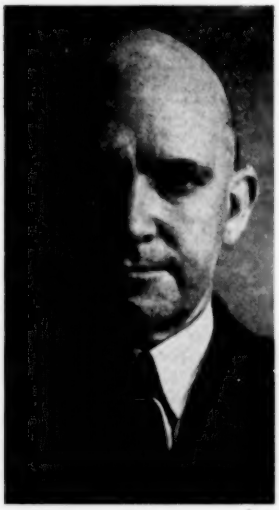
The following candidates have completed all the requirements of the Sixth Professional Examination for the degree of M.B., B.Ch. in the summer examinations, 1955:

Abrahams, C.
Abramowitz, I.
Adler, H. D.
Alsfine, H.
Bader, S. J.
Beck, J. M.
Blecher, T. E.
Boorany, S. E.
Brueckner, R. M.
Chesler, E.
Cohen, A. D.
Cohen, M. L.
Curlewis, C. C.
Ebrahim, I.
Eidelman, S.
Engelbrecht, P. J.
Farrell, V. J. R.
Ferguson, N. M.
Freeman, M.
Geffen, H.
Gentin, B.
Goldstein, D. J.
Gruebel Lee, D. M.
Gruebel Lee, E. C.
Haffajee, D.
Hassim, A.
Ibach, E. G.
Ipp, H.
Jabour, C.
Johnson, R. P.
Josman, H.
Krikler, R.
Krohn, J.
Kulwinsky, I.
Lebanon, E.
Libesman, R.
Liknaitzky, D. G.
Marburg, L.
McGregor, J.
Meskin, D. J.
O'Donovan, T. G.
Pienaar, B. T.
Pillay, U.
Rabinowitz, B.
Roman, H. E. H.
Rothschild, E. E. A.
Safro, I. L.
Saperson, D.
Schiller, C.
Schneider-Kuper, E.
Schwartz, N. L.
Seimon, L. P.
Shenker, C.
Shnier, J.
Sindelman, I. L.
Slone, D.
Steffensen, E. B.
Strasburg, E. R.
van der Walt, H. J.
Wannenburg, P. J. D.

IN MEMORIAM

MARCUS COLE ROUS, F.R.C.S.

Professor J. H. Louw, of Cape Town, writes: The sudden death on 18 November of Dr. Marcus Cole Rous at the age of 52 years removes a leading figure in South African surgery and a man greatly admired by the profession.



Marcus Cole Rous, F.R.C.S.
Photo : Cape Times

During his active public life Cole Rous filled many positions of trust and influence in the profession. For many years he was a member of the Council of the Cape Western Branch of the Medical Association of South Africa, and at the time of his death he was President of the Branch and member of Federal Council and the Head Office and Journal Committee. A great impression was made by his Congress Public Lecture, which he delivered in September 1952 at the South African Medical Congress at Johannesburg. He was an active member of the teaching staff of the University of Cape Town for nearly 20 years, and held the Chair of Surgery during 1947-48. Unfortunately he was compelled to relinquish the post on account of ill health. He played a prominent role in the establishment of the new College of Physicians and Surgeons of South Africa, and was vice-chairman of the steering committee.

Marcus Cole Rous was not only a surgeon of outstanding ability, but also one of the best teachers of surgery that this country has seen. He had the ability of simplifying problems and imparting knowledge to others in such a way that the lessons taught were never forgotten. Clinical observations were usually illustrated by anecdotes derived from his experience, and his numerous quips are still being repeated years after they were related. He has certainly left his stamp on many Cape Town graduates, and has contributed a great deal to their own success and the development of surgery in South Africa.

Cole Rous was a deep thinker and a man who had many and varied interests outside his own professional sphere. He was a keen student of philosophy and psychology as well as of the classics, music and art. His hobbies included flying, yachting, fishing and carpentry, each of which he pursued with such enthusiasm and tenacity of purpose that he became an authority on them all. He certainly had a phenomenal brain, and could adapt himself to almost everything.

To those of us who knew him well the one thing that made Marcus a really great man was his courage. He was never afraid to stand up for his rights and for those of others, but openly spoke his mind. The admirable manner in which he accepted his illness and continued to be an active member of the profession bears testimony to his courageous spirit.

Marcus will be greatly missed by many, and his influence will continue to live. It is indeed a privilege and an honour to have been a close associate of his.

PASSING EVENTS : IN DIE VERBYGAAN

Royal College of Physicians of Edinburgh. At a Quarterly Meeting of the College held on 1 November 1955, the President, Sir Stanley Davidson, in the chair, the following were elected Members of the College: A. B. Murray, B.Sc., M.B. (Cape Town); R. G. Hendrickse, M.B. (Cape Town); S. Axelrod, M.B. (Witwatersrand); R. F. Fouché, M.R.C.S. (Eng.), L.R.C.P. (Lond.).

* * *

Mr. B. W. Franklin Bishop, F.R.C.S. (Edin.) wishes to inform his colleagues that he has commenced practice as a Plastic and Facio-Maxillary Surgeon at 711 Payne's Buildings, West Street, Durban. During his 2½ years in the United Kingdom, Mr. Bishop was assistant to the Nuffield Professor of Plastic Surgery (Professor T. P. Kelner) at the Churchill Hospital, Oxford. Mr. Bishop has been appointed Visiting Plastic and Facio-Maxillary Surgeon, Addington and Wentworth Hospitals, Durban.

* * *

Dr. N. Sapeika, of the Department of Pharmacology of the University of Cape Town, has been appointed Associate Professor of Pharmacology as from April 1956. Professor Sapeika has accepted an invitation to lecture with the status of Associate Professor at the Medical School, Jerusalem, from January to July 1956, and will be leaving shortly.

Dr. W. Norman Taylor, formerly Professor of Hygiene at the University College of Fort Hare, and for the past 2 years on the staff of the International Labour Office, Geneva, has transferred to the World Health Organization as a consultant on education and training programmes. He recently passed through the Union on his way to Nigeria, where he will advise the Government on the training of auxiliary health workers.

* * *

At the Annual General Meeting of the *South African Orthopaedic Association*, held in Pretoria on 19 October 1955, Sir Reginald Watson-Jones, London, and Professor Bryan McFarland, Liverpool, were elected honorary members of that Association.

The following office-bearers were elected for the ensuing two years: *President*—Mr. C. T. Moller; *Treasurer*—Mr. W. T. Ross; *Secretary*—Mr. Sidney Sacks; *Committee Members*—Messrs. J. M. Edelstein, G. T. du Toit, J. G. du Toit and G. Dommisse.

* * *

The Professional Provident Society of South Africa. This Society is opening its own offices with effect from 1 December 1955. The address will be 1003 Cavendish Chambers, Jeppe Street, Johannesburg. The postal address, P.O. Box 6268, Johannesburg, will remain the same.

CORRESPONDENCE : BRIEWERUBRIEK

CEREBRAL PALSY SCHOOLS

To the Editor: We have read with interest the letter[1] in your issue of 8 October dealing with private cerebral palsy schools.

I should like to point out that in Natal treatment centres for the cerebral palsied child have been in existence for many years; and started before the Forest Town School was opened. I refer to:

(*a*) *The Open Air Government School, Durban.* Day scholars suffering from cerebral palsy have been admitted since 1921, and received medical treatment at the school. Since 1952 cerebral palsy cases from all over Natal have been admitted to the hostel attached to the school. This school admits children suffering from all types of physical handicaps.

(*b*) *The Uplands Government School, Swartkopskloof.* Cerebral palsy cases from all over Natal have been admitted since 1946

from the Uplands Orthopaedic Home, where they receive treatment. This home admits children suffering from various orthopaedic handicaps.

(*c*) *Itinerant Teacher for Cerebral Palsied.* In Durban a number of cerebral palsy cases who are not eligible for admission to existing institutions are given tuition by an itinerant teacher, and receive treatment at the Cerebral Palsy Clinic at Addington Hospital.

I shall be glad if you will publish this letter in order to correct the wrong impression which may have been given by the penultimate paragraph of the letter mentioned above.

E. Beardmore
Chairman
Natal Provincial Cripples Care Association

Uplands Orthopaedic Home
Swartkopskloof
Natal
14 November 1955

1. Vorwerg, F. M. T. and Levitt, S. (1955): S. Afr. Med. J., **29**, 976.

CARDIAC ARREST DURING SURGERY

To the Editor: Dr. Katz[1] has written an excellent article on this subject, but he states that 'the abdomen should never be opened primarily to initiate (cardiac) massage'. I consider this to be a very dangerous and harmful statement.

I am quite aware that the transthoracic approach is the best one, but I think Dr. Katz perhaps does not know of the many small hospitals and nursing homes in the Union, where cardiac arrest may occur and massage have to be undertaken, perhaps by a houseman (or even by the anaesthetist, if the cardiac arrest occurs during a dental procedure), leaving a nurse to respire the patient with oxygen. The thoracic approach is not as easy as Dr. Katz makes out in all cases; I have attempted it myself during an operation for resection of the oesophagus, and it was impossible in this case to get my hand into the chest before a rib-retractor was inserted.

A rib-retractor is not always available in every small hospital or nursing home, and, for people who are not familiar with its use, the time taken to get it into position would exceed the 3 minutes available to restore circulation.

A scalpel is always available, and one slash through the abdominal wall will enable the heart to be palpated through the diaphragm; in my opinion this will give a far greater chance of survival in the majority of cases outside the departments of general and thoracic surgery of the teaching hospitals.

J. D. M. Barton

31 Yalta Road
Pietermaritzburg
16 November 1955

1. Katz, G. (1955): S. Afr. Med. J., **29**, 1065 (12 November).

BLADDER DRAINAGE

To the Editor: May I be permitted to comment on Mr. Retief's article[1] on bladder drainage by means of perineal urethrostomy? This method was advocated and popularized by Mr. Sandrey of St. Peter's Hospital, London, for many years, with excellent results.

With regard to the technique of the operation, I should like to describe a simplified method which I have used on several occasions, on bad-risk cases. This method can be used, if necessary, without moving the patient from his bed.

The procedure is carried out under local anaesthesia. The perineum just behind the scrotum is infiltrated with novocaine. The urethra itself is filled with local anaesthetic jelly, and a catheter is introduced and is pushed in as far as it will go, until only the end is visible. This end is grasped with a slender curved artery-forceps. The forceps and catheter are pushed into the urethra until the tip of the forceps can be felt in the perineum just behind the scrotum and a small incision is made over the palpated tip of the forceps. The catheter is then pushed out into the perineum and the forceps withdrawn. The catheter is anchored with a stitch after being adjusted at the correct level. A Foley catheter can be used in the same way, provided it is not more than an F18, and the secondary channel is trimmed down so as to be less bulky.

It is important to have the urethrostomy just behind the scrotum, as if it is too far back the patient is liable to sit on the tube, with undesirable results.

This whole procedure takes a couple of minutes in all, and does not appear to upset the bad-risk case at all.

I. V. Rogoff

1006 Medical Centre
Jeppe Street
Johannesburg
17 November 1955

1. Retief, P. J. M. (1955): S. Afr. Med. J., **29**, 1074.

SPECIALIST, CONSULTANT AND GENERAL PRACTITIONER

To the Editor: I wonder whether it is possible to look at this question of Specialist, Consultant and General Practitioner from a different angle.

As a specialist I find that about 80% of my patients are being referred to me by general practitioners and other specialists, and 20% come in of their own accord, having been recommended to see me by a friend of theirs, and it is this 20% which will be affected by the proposed consultants' register.

With the 20% it is often found that they have been attended by their general practitioners, who have persevered with one treatment or another without improvement until the patients have lost confidence in them. Often the patient has gone from one G.P. to another, and still has not been referred for specialist opinion. These are the patients who often seek advice and obtain relief from unqualified practitioners, and by adopting a compulsory consultants' register we shall be encouraging them to seek the aid of the quacks instead of the regular medical specialists. I, for one, do not believe that any patient will prefer to visit a specialist on his own, and pay the extra fees involved, if he can get the same treatment from his G.P. at a much lower fee. This is surely a question of supply and demand.

Another pertinent question is whether the general practitioners have the legal or the moral right to demand that all specialists should be consultants only. What an outcry there would be by the G.P.s if the specialists demanded that the G.P. should not perform any specialized service for his patient when there is a specialist available! We all know the surgeon G.P., the radiologist G.P. and the physiotherapist G.P. We remember the rush to buy ultrasonic machines a few years ago by all and sundry. Diathermy machines are common equipment in the rooms of many G.P.s even in the larger centres, where there are specialists available to do the work.

The crux of the matter is that the public today are becoming educated, or at least they imagine they are, and it is *their* demands that have to be met by the medical profession.

18 November 1955 *Epicure*

'MONGOLISM' IN THE BANTU

To the Editor: Recently there have appeared in the *Journal* several articles on Mongolism in the Bantu, the latest of which is contributed by Dr. B. J. Kaplan.[1] As a few of these cases have hitherto been reported in the South African literature, it is assumed that it is a very rare condition and therefore the report of an isolated case is justifiable.

I would like to refute this assumption on the grounds of my own experience and that of several others who in the course of their work come in contact with large numbers of Bantu children. Luder and Musoke[2] report 5 cases seen in the neighbourhood of Kampala, Uganda. They make the point that the only accurate method of estimating the incidence of mongolism is by surveys amongst newborn babies and infants. E. Kahn of Baragwanath Hospital, in a personal communication to these authors, stated that about 3 cases per annum are diagnosed in his Unit.

At my out-patient clinics at Edenvale Hospital, Johannesburg, I personally see at least one case per year. These children are

brought not on account of their mental state, but for other illnesses such as malnutrition, gastro-enteritis, or respiratory diseases. Edenvale Hospital draws cases from Alexandra Township, which is a large Native township in the neighbourhood of Johannesburg. We treat, however, only sick children in our hospital, and therefore see a relatively small proportion of that population.

Whilst I agree that the racial incidence may be very important in the etiology of certain diseases, I do not think that the report of one case is sufficient on which to make assumptions. Mongolism in the Bantu is well worthy of study. If Dr. Kaplan were to choose as a sample a sufficiently large Native area in South Africa in which to investigate the incidence of mongolism, he would be able to establish or refute the assumptions that he has made in his article. In order to do this effectively, however, he must have the opportunity to study all the infants in their first year of life. He must also enlist the cooperation of other doctors, midwives and nurses working in that area.

Luder and Musoke state that amongst their cases the age-incidence of mothers of mongols is lower than that reported by authors describing the incidence in Western communities.

In conclusion I would like to quote an incident at which I was present at the Postgraduate Medical School of London. A mongol was being demonstrated and amongst the signs stressed was a single transverse crease of the hand. This was considered to be an important diagnostic point. At the end of the demonstration a Chinese doctor, who already had passed her M.R.C.P. and F.R.C.S., put out her hand to the lecturer to show that she had a single transverse palmar crease. Obvious inferences can at times be refuted by the exception.

Ben Epstein

104 van Riebeek Medical Buildings
Schoeman Street
Pretoria
21 November 1955

1. Kaplan, B. J. (1955): S. Afr. Med. J., **29**, 1041 (5 November).
2. Luder, J. and Musoke, L. K. (1955): Arch. Dis. Childh., **30**, 310.

NOTES ON DISRUPTION

To the Editor: As usual I very much enjoyed reading Mr. George Sacks's latest contribution to surgery. His article *Notes on Disruption* not only draws attention to this too frequent and distressing complication which faces the surgeon after any extensive laparotomy but is written in his usual style which makes his writings literary gems.

I entirely agree with him as regards the causes of disruption, and they should be borne in mind at the beginning of every abdominal closure and afterwards.

Acting on these principles I have been fortunate enough to have had no disruptions in a series of over 4,000 major abdominal operations because:

1. The Lord was good to the patients.
2. The main fascial layers are *always* closed with interrupted sutures—even in the direst emergency when speed is the deciding factor. An interrupted suture, here more than ever, may be the stitch in time that saves nine at a later date.
3. In *all* cases of malignancy the majority of the sutures used in closure are of unabsorbable material—surgilon, silk or fine 'Staybrite' steel wire.
4. I always use several stay-sutures of dermalon in the form of a figure-of-eight with the protruding ends tied over a gauze 'sausage'. The advantage is that with this type of suture the more the patient strains the more the suture approximates the fascial layers, since the figure-of-eight pulls in opposite directions. The gauze sausage on the other hand exerts firm uniform pressure over the entire length of the wound, thus keeping it dry. Oozing keeps the wound edges apart, thus delaying healing and favouring infection, which in turn predisposes to disruption. Warning: These figure-of-eight stitches are sometimes rather painful and difficult to remove; this operation had therefore best be left to the sister, lest the surgeon's popularity with the patient be decreased.
5. Protein and vitamin-C deficiency are prevented because all the bigger cases on post-operative intravenous fluids are started off, as a routine, by glucose in saline, followed by blood and amigen, depending on the nature of the operation. Amigen and vitamin C, however, are given as a matter of course.
6. The anaesthetist must be watched, for in spite of the increased magnitude of present-day operations made possible by better anaesthesia, he can still be a menace to both the patient's life and the surgeon's reputation. Beware of the super-specialist who completely knocks out the patient and keeps him on artificial respiration till it is dangerous to do so any longer and the surgeon consequently has to close up the abdomen of a patient fighting for his life and straining his abdominal muscles in the process. This is unfortunately experienced too often these days when anaesthesia consists of premedication followed by abolition of consciousness, induction of paralysis, and finally resuscitation, in that order. Close cooperation, therefore, between the surgeon and the anaesthetist is one of the main prophylactic measures against disruption, and with gratitude I pay tribute to my anaesthetist, who in no small way contributed to the small measure of success I have had in preventing dehiscence after operations.

Finally, Mr. Editor, may I add that if your excellent journal published more of these practical 'bread-and-butter' articles like Mr Sacks's, it would enjoy an even greater measure of popularity than it does at present—if that were possible.

Willem P. Steenkamp, Jnr.

S.A. Mutual Building
Cape Town
23 November 1955

1. Sacks, G. (1955): S. Afr. Med. J., **29**, 1081 (19 November).

WOUND RETRACTION IN OPERATION ON THE BILIARY TRACTS

To the Editor: I enclose a description and sketch of a simple apparatus for use in operations on the biliary tracts, to aid in wound retraction.

It may interest those surgeons who, owing to lack of sufficient staff, have had to do these operations with only a single assistant.

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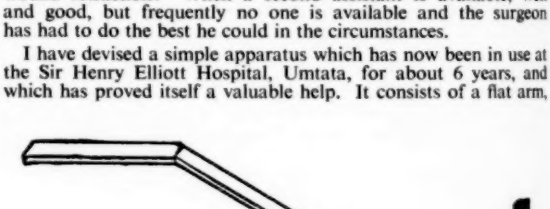
THE APPARATUS

How often has it proved necessary to have a second assistant during operations on the biliary tracts in order to obtain efficient wound retraction! When a second assistant is available, well and good, but frequently no one is available and the surgeon has had to do the best he could in the circumstances.

I have devised a simple apparatus which has now been in use at the Sir Henry Elliott Hospital, Umtata, for about 6 years, and which has proved itself a valuable help. It consists of a flat arm,



3 feet long, bent at an angle of 45° 12 inches from one end. This end fits into the standard clamp which normally holds the shoulder-supports on the operating table. To the other end is soldered a vertical standard 12 inches in height, notched at intervals.

When the arm is in position on the operating table, the retractor is applied to the wound and attached by means of a fine chain to the vertical support on the arm. The position of the retractor can be adjusted by sliding the table clamp along its rail.

Since the arm is relatively long it possesses some elasticity and a rigid pull is not, therefore, exerted on the edge of the wound. Very adequate retraction is maintained throughout the operation.

The apparatus is intended to aid the retraction of the *right* wound-edge. A sketch is appended.